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CLINICAL EXPERIENCES IN SURGICAL TREATMENT OF FACIAL PALSY BY AUTOPLASTIC NERVE GRAFTS

THE BALLANCE-DUEL METHOD

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At the meeting of the American Otological Society in May, 1931, Sir Charles Ballance of London, England, and I reported the results of a year's research on facial palsy, consisting of surgical experiments on baboons, monkeys, cats and frogs. We had aimed to evolve a method of restoring lost facial function which would eliminate associated movements. Cures hitherto effected by means of an anastomosis of the facial nerve with an adjacent nerve in the neck, such as the spinal accessory, the hypoglossal, the descendens noni and the glossopharyngeal, had invariably been marred by associated movements.

Our experiments were carried out at my country place, where an experimental animal laboratory had been built for the purpose. There, the quiet environment, pure air, sunshine, protection from drafts, cleanliness, careful feeding and kindly treatment by trained attendants insured us against the vitiation of an experiment by the untimely death of any of the animals.

A number of experiments and observations are still being carried out on animals which have been in captivity for more than two years.

The last day of the meeting of the American Otological Society, in May, 1931, was given over to a visit to the animal laboratory at Laurelwood, where the members, and many invited guests, were shown sixty or more animals on which operations had been performed, exhibiting various phases of our experiments with anastomosis of the nerve and autoplastic nerve grafts.

In the subsequent four months further experiments were made on more than fifty animals. The complete data, including the final experiments, were published in the January, 1932, issue of the ARCHIVES OF OTOLARYNGOLOGY. A reprinted monograph has been sent to all the members of the society.

Read at the Thirty-Eighth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City, N. J., June 23, 24 and 25, 1932.

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Sir Charles Ballance returned to London last September with one hundred and fifty anatomic specimens, the preparation of which—for microscopic study and report—he has supervised at the Royal College of Surgeons. The histologic and pathologic report will be published within a year.

The practical outcome of our work, so far as it immediately interests otologists, lies in the fact that our experiments led us to deprecate the anastomosis of the facial nerve with one of the adjacent nerves in the neck as the method of restoring lost facial function, and to advise, in place of this method, the employment of an autoplastic graft to bridge the gap from the proximal to the distal segment caused by injury or disease.

The use of autoplastic grafts for the restoration of function in other nerves has been successfully carried out for many years. Létiévant,[1] as early as 1873, advocated the plan. In our recent monograph [2] we called attention to various intratemporal operations by Stacke [3] in 1903, Alt [4] in 1908, Sydenham [5] in 1909, Marsh [6] in 1909, Ney [7] in 1922 and Martin [8] in 1931, showing ingenious methods of dealing with the facial nerve. In 1927, Bunnell,[9] of San Francisco—without having any knowledge of Ney's work—having had a wide experience in nerve grafting in other regions, reported an intratemporal repair of a facial nerve done in 1925. Although he did not employ a graft in that case, he suggested that, where the gap was wide enough to require it, a graft from the sural nerve might be employed. This he successfully did in 1930, using a branched graft from the sural nerve, 1½ inches (3.77 cm.) in length.[10]

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1. Létiévant: Traité du section nerveuse, Paris, J. B. Baillière et fils, 1873, p. 44.

2. Ballance, C., and Duel, A. B.: The Operative Treatment of Facial Palsy by Introduction of Nerve Grafts into the Fallopian Canal and by Other Intratemporal Methods, Arch. Otolaryng. **15**:1 (Jan.) 1932.

3. Stacke, quoted by Alt.[4]

4. Alt, F.: Verhandl. d. deutsch. otol. Gesellsch., 1908, p. 191.

5. Sydenham: Brit. M. J. **1**:1113, 1909.

6. Marsh: Brit. M. J. **1**:1356, 1909.

7. Ney, K. W.: Facial Paralysis and the Surgical Repair of the Facial Nerve, Laryngoscope **32**:327, 1922.

8. Martin, R. C.: Intratemporal Suture of Facial Nerve, Arch. Otolaryng. **13**:259 (Feb.) 1931.

9. Bunnell, S.: Suture of the Facial Nerve Within the Temporal Bone, Surg., Gynec. & Obst. **45**:7, 1927.

10. Unfortunately, Sir Charles and I were not aware of this work at the time our monograph was written. It has only come to my notice, since the presentation at Atlantic City, in a letter to Sir Charles which arrived after our return from the meeting of the American Laryngological, Rhinological and Otological Society. I am glad to have it in time to include it in this publication.

The details of the experiments which eventually led us to this conclusion, together with the technic of the method which proved invariably successful in twenty-four animals, appeared in the published account of our work.

I must pause, at this juncture, to reiterate what I have said on many previous occasions, viz.: that but for Sir Charles' clear vision and amazing energy this could not have been accomplished. It has been a great joy to me to have been associated with him, and to have aided in bringing it to a successful conclusion. To express adequately in words the inspiration he has been to me, or to tell fully all the knowledge he has so generously passed on to me from his long and rich experience, is quite beyond my power.

I now wish to report briefly twelve cases—one double—of facial palsy in human beings who have been operated on according to the principles evolved from our animal experiments.

REPORT OF CASES

CASE 1.—A girl, aged 8½ months, had a purulent discharge of the left ear accompanied by a postauricular subperiosteal abscess. Immediate complete facial palsy on the left side followed operation on March 16, 1931. Exploratory operation was done on March 18. The stump of the divided nerve was found in the neck at the posterior border of the parotid gland. A faradic current applied to this stump caused contraction of all the muscles of the left side of the face. The end of the proximal segment was found at the level of the horizontal semicircular canal. The ossicles and drum were removed and the facial nerve uncovered to the geniculate ganglion (the measured distance of the gap from the proximal to the distal segments was 27 mm.). An autoplastic graft from the external respiratory nerve of Bell was laid in a gutter in the temporal bone from the proximal end, without suture, to the distal end, which was sutured by one strand of finest silk attached to a needle without a knot (special suture made by Davis and Geck).

CASE 2.—Mrs. B., aged 34, had chronic otorrhea; she underwent a radical operation on the mastoid on July 28, 1930. Immediate complete facial palsy developed. There was no faradic response but a slight response in the muscles to galvanic positive closure. The patient was referred by Dr. Marvin F. Jones from his clinic at the New York Post-Graduate Hospital, where she had presented herself long after the injury. Exploration eleven months after injury, June 25, 1931, revealed a mangled facial nerve which had been replaced by granulation and fiber tissue. The measured gap, after this was removed, was 30 mm. in length. This gap was bridged by the external respiratory nerve of Bell, laid in from the geniculate ganglion to 6 mm. outside the stylomastoid foramen. About three months later—the graft having lived and been covered in with a deep bed of healthy granulations—a plastic operation was done to close up the posterior wound with a less disfiguring scar. Advantage was taken of the anesthesia to try the effect of a powerful faradic current over the graft at the angle of the jaw. All the muscles of the face responded. No spontaneous movements were visible, however, until about eight months after operation, when it was noticed that the patient closed

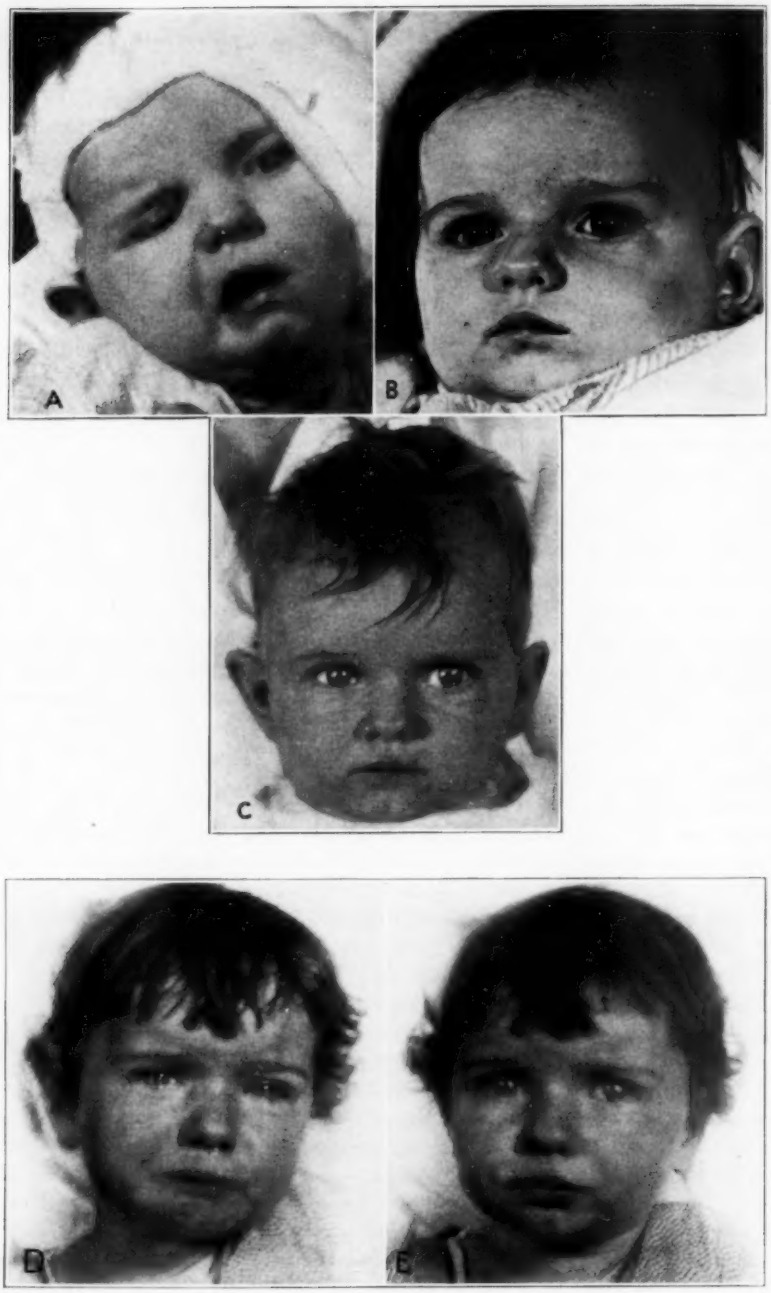

Fig. 1 (case 1).—*A*, complete palsy of the face showing the eye wide open and the mouth distorted; *B* (sixty-one days), slight spontaneous movement of the orbicularis oris and levator labii superioris alaeque nasi; (seventy days), spontaneous movements of the muscles around the eye, wing of the nose and the mouth, caused by tickling of the face with a string during sleep; partially closed eye; *C* (one hundred and one days), definite response in all the muscles to the faradic current; spontaneous use of the muscles; closed eye tightly in sleep and on conjunctival irritation; face symmetrical in repose; *D* and *E* (fourteen months), face symmetrical in action and repose; synchronous wink—normally and on irritation; emotional contraction—crying.

her eye in sleep and was able to make a slight motion, on exaggerated effort, with the orbicularis palpebrarum. The face has gradually pulled around to the present noticeable state of improvement. She is beginning to use the muscles around the eye and mouth. Moving-pictures which have been made show the rapid recent improvement better than the still-pictures. It seems that, when improvement begins, its subsequent progress is rapid. She is able to move all muscles although imperfectly. She has a plate with artificial teeth (made by Dr. J. J. Martin of New York) with an ingenious arrangement for improving the appearance in repose.

Fig. 2 (case 2).—*A* and *B*, before nerve graft, June 25, 1931, with face in repose and smiling; *C* and *D*, in repose and action, May 15, 1932.

Case 3.—Mr. F., aged 28, had complete facial palsy on the left side immediately following an operation for removal of exostosis of the temporal bone; there was also loss of hearing and static sense on that side. Thirty-one millimeters of intercostohumeral nerve replaced the absent nerve from the geniculate ganglion to 7 mm. outside of the stylomastoid foramen. This graft was not successful (necrosis), which is the only failure of a nerve graft to live in this series. A double intercostal graft three weeks later lived. There is no return of faradic response as yet; the patient has had some feeling of formication in the face; he can close his eye and hold it closed. It is too early as yet to tell anything very definite in a case of two years and two months' standing.

CASE 4.—Miss S., aged 21, had suffered from Bell's palsy on the left side for ten years with partial recovery. Bell's palsy was present on the right side for three and one-half months with partial recovery beginning after two months. Both sides showed a slight response to the faradic current. With both sides partially recovered and nearly equal, the patient had a most peculiarly solemn look. On Nov. 14, 1931, after ten years, she suddenly had a second complete Bell's palsy. Faradic response continued for four days and then disappeared; galvanic reaction of degeneration was obtained on the left. On Dec. 1, 1931, operation was performed—decompression of both facial nerves from the stylomastoid foramen to the level of the horizontal semicircular canal. On April 15, 1932, both sides of the face moved equally and synchronously, either by voluntary effort or emotionally.

All cases, except nos. 1, 2 and 4, are reported and a short history given merely to show the variety and extent of injuries in which nerve

Fig. 3 (case 3).—Before nerve graft, Oct. 16, 1931.

grafts have been employed. The grafts have lived in all, and, in many, have already given promise of recoveries which I hope to report in another year.

CASE 5.—Miss O., aged 19 (referred by Dr. Edmund P. Fowler from his clinic at the Manhattan Eye, Ear and Throat Hospital), at 5 months of age underwent an operation on the mastoid followed by complete facial palsy. This was of eighteen years' duration. There was no faradic response and slight response to galvanic positive closure. Operation was performed on Dec. 3, 1931. The distal segment was found curled up on the posterior surface of the parotid gland; the proximal segment was found just below the geniculate ganglion. Thirty millimeters of intercostal nerve replaced the gap. It is too early after operation to predict the result; however, there is a returning reaction to the faradic current. The patient has a feeling of formication in the face at times. There is great promise of improvement, although probably it will never be complete after such a long period of disuse.

Fig. 4 (case 4).—*A* and *B*, just before decompression, Dec. 1, 1931; *C* and *D*, five and one-half months later.

Fig. 5 (case 5).—*A*, in action before operation; *B*, in repose before operation.

CASE 6.—Mr. M., aged 28, underwent an operation in Russia thirteen years before the present operation, with complete paralysis of the left side ever since. There was only slight galvanic response—10 milliamperes, positive closure—while under anesthesia. Thirty-four millimeters of intercostal nerve replaced the destroyed segment. At three and one-half months the patient began to feel formication in the muscles around the mouth; with exaggerated effort he could close his eye and hold it closed. There is good promise, in a year or two, of a much improved face in repose, closure of the eye and partial movements of the face.

Fig. 6 (case 6).—Before operation, Dec. 29, 1931.

Fig. 7 (case 7).—Before operation, Jan. 28, 1932.

CASE 7.—Mr. C., aged 34, underwent an operation for removal of a parotid tumor eight years before the present operation. All branches of the facial nerve were divided. Operation was performed on Jan. 28, 1932. The proximal segment was found at the exit from the stylomastoid foramen; the parietal branch was found on the ramus of the jaw, 40 mm. of intercostal nerve graft being sutured to the stump extending to this branch; the temporal branch was found near the zygomatic arch, 20 mm. of intercostal nerve graft from the main stump being sutured to this branch. Eventual partial recovery is expected from the fact that the patient has "felt a sensation of movement" repeatedly and is beginning to get slight response to faradic stimulation.

CASE 8.—Miss C. C., aged 15, had paralysis one year following injury in a simple operation on the mastoid. On Feb. 25, 1932, 20 mm. of intercostal nerve was grafted; the graft has lived. It is too early for any evident response.

Fig. 8 (case 8).—Before operation, Feb. 25, 1932.

Fig. 9 (case 9).—Before operation, March 9, 1932.

CASE 9.—Miss W. S., aged 12, had paralysis seven weeks following injury in a simple operation on the mastoid. On March 9, 1932, 16 mm. of intercostal nerve was grafted, two strands being used.

Case 10.—Mary A., aged 3, underwent a secondary operation on the mastoid, on March 8, 1932, followed by immediate facial palsy. An exploratory operation was done on March 10. A depressed fracture of the fallopian canal was found in the vertical portion. The facial nerve was uncovered above and below the site of injury for 5 mm. Following the decompression perfect recovery is anticipated.

Case 11.—Charles J., aged 7, had a depressed fracture of the fallopian canal for three years with partial palsy only. On April 9, 1932, there was faradic response. Decompression was achieved by removal of the fractured segment. It is too early to predict results after compression for three years. If this had been done immediately after injury, as in case 10, it would probably have resulted in perfect cure.

Fig. 10 (case 10).—*A*, before decompression, March 10, 1932; *B* and *C*, in repose and action, three months later.

Case 12.—Mary Y., aged 12, suffered injury of the facial nerve by a ligature passed deep in the neck to check arterial hemorrhage during removal of a cystic tumor in the parotid region. She came under my care ten days later. On April 29, 1932, there was no faradic response; with galvanic positive closure, there was response in all muscles. Dissection of the facial nerve showed the main branch just outside the stylomastoid foramen constricted by the ligature. Seven millimeters of necrotic nerve was removed and replaced by two strands of the seventh intercostal nerve. The nerve grafts lived. Plastic closure was done after three weeks. Perfect recovery is expected.

Fig. 11 (case 11).—Before decompression, April 9, 1932.

Fig. 12 (case 12).—Before operation, April 29, 1932.

COMMENT

What has been learned from these experiences?

1. It is too early, in many of the cases, to predict how complete a recovery will take place. They are recorded to show the variety and extent of injury, and the final outcome will be reported at a later date.

2. It is certain that in all these operations the gap, caused by removal of the injured area, has been replaced by a living autoplastic nerve graft which conveys impulses from the cortical center in the brain to the peripheral endings of the facial nerve. Naturally, there can be no associated movements.

3. The experience of actually seeing the extent of the injury in so many cases of facial palsy following operation on the mastoid has been illuminating. Surgeons who have repaired such injuries by anastomosis with adjacent nerves in the neck have had no reason to examine the seat of injury. To them the amount of damage suffered in nearly all the cases in this series, as disclosed by opening the fallopian canal from the stylomastoid foramen to the geniculate ganglion, will doubtless be astonishing. Occasionally, an operator who has had an accident may be aware of what has happened. I believe that in most cases the operator has no accurate knowledge of how, when or to what extent the nerve has been damaged.

Of the twelve cases here reported, in one there was a double Bell's palsy. Of the eleven accidental injuries, two were slight and required only the removal of a depressed fracture of the external wall of the fallopian canal over a small area and decompression above and below the site of injury. In the remaining nine the area of destruction of the nerve varied from 15 to 40 mm. in length. These wide gaps were a determining factor in the selection of the particular nerve from which the graft was taken.

4. It seems certain that even most careful observation of the face, by the anesthetist during the operation, for sudden spasm of the muscles as an indication of injury of the nerve is unreliable. As pointed out in our recent monograph, trauma severe enough to cause facial palsy may be inflicted without any observed spasm; and, while it may be informative at times, when positively seen, lack of such observation is really no accurate means of knowing whether, when or how extensive an injury to the facial nerve may have occurred.

5. These experiences also point conclusively to the advisability of uncovering the nerve at once, whenever facial palsy immediately follows an operation on the mastoid, in order to determine the extent of the damage. The rewards of such action are manifest. Compression or slight injuries may then be remedied by decompression—with assurance that there will be complete, or nearly complete, recovery in many

cases—where, on the other hand, long delay, bolstered up by vain hope, will often result most unsatisfactorily.[11]

In addition to such an occasional case, there will be many in which prompt inspection will disclose the fact that the accident has destroyed or damaged a longer segment of nerve. Immediate operation will permit a decompression of the nerve, above or below the point of injury, in time to avert the dire consequences of prolonged inflammatory compression. A suitable graft may be introduced to replace the damaged segment at once. There can be only slight atrophy of the muscles from nonuse, and, consequently, a quick and more perfect recovery is assured.

The great advantage of being able to identify the nerve in the neck by the faradic current, before the reaction of degeneration has taken place, should also be considered. This probably does not last more than seventy-two hours; in the four monkeys, in which, after division, the reaction was tried at twelve hour intervals, it disappeared in from sixty to seventy-two hours. It was of inestimable value in finding the facial trunk, severed close to the posterior border of the parotid gland, in case 1, in which operation was done the second day after injury.

6. The experience with the double Bell's palsy bears out the contention of Sir Charles most forcibly, that decompression should be done in these cases when, after six or eight weeks' observation, only partial recovery has taken place and is apparently at a standstill. Miss S. (case 4), when first seen by us, gave a history of Bell's palsy on the left side of ten years' duration, followed by slow and incomplete recovery. Two months before, Bell's palsy had suddenly developed on the right side. This had just begun to show partial recovery. The result was that, while there was a sluggish movement of both sides, on great effort, there was a peculiar solemnity to the patient's whole expression, which is beyond description. Nevertheless, with both sides partially paralyzed, and nearly equally, the face appeared less grotesque—both in repose, and when she attempted to use the muscles—than when one side only had been at one time partially, and, at another time, completely paralyzed. The face was fairly symmetrical, though peculiarly solemn.

A double decompression was advised. This she refused, or rather deferred. In the meantime she came at short intervals for observation. On Nov. 16, 1931, she returned with the history that on November 14, following some slight exposure to cold (as indefinite as that which had caused her two previous attacks), the side on which there had been semiparalysis for ten years had become completely paralyzed again. The electric reactions of wallerian degeneration took place. It is interesting to note that for seventy-two hours following complete palsy, there was a markedly increased response to faradic excitation; after

11. Ballance and Duel (footnote 2, p. 29).

seventy-two hours there was a typical loss of response to faradic, and quick response to galvanic positive closure.

After two weeks, without any apparent recovery, the patient consented to a double decompression. Both nerves were successively uncovered from the stylomastoid foramen to the horizontal semicircular canal. An amazing thing to witness was that, on each side, as the sheath was slit open, the nerve swelled immediately to twice its former size. The phenomenon might be likened to what one might expect if a gum-elastic cylinder, ⅛ inch (3.17 mm.) in diameter, were compressed or drawn in to a cloth tube 1/16 inch (1.58 mm.) in diameter. On slitting the cloth tube, the rubber, relieved from pressure, would immediately expand to double the compressed size.

Despite the slitting of the sheath over 16 mm. of its length, the left partially recovered nerve did not go through the reaction of degeneration. Faradic response was always present. The right nerve, as I have just said, was completely degenerated at the time of decompression. Both have now recovered, about equally, to a condition far superior to the best that either had ever shown in their partial recovery from the initial Bell's palsy.

As you have seen, the patient now moves all muscles of both sides fairly synchronously, except the frontalis on the left. Even this is improving. Improvement, slow in starting, has since advanced rapidly. I shall not venture to say how complete a recovery will take place, but I am sure that this patient is already glad that she finally consented to undergo the ordeal of an operation, recommended only from theoretical conclusions based on animal experiments.

7. We definitely demonstrated, in our animal experiments, that any autoplastic nerve graft—either motor or sensory, with the direction of proximal and distal ends either maintained or reversed, will successfully bridge the gap and restore the function of a divided facial nerve. We employed reversed and unreversed facial, intercostal, descendens noni, great auricular, musculocutaneous and external respiratory (nerve of Bell) nerves, with invariable success. Various lengths were used, from 2 up to 16 mm.

In most of the experiments proximal and distal relations were preserved. The rapidity of recovery bore some relation to the length of the graft, i. e., the shorter the graft, the quicker the regeneration.

In the later experiments we did a great many repairs of varying lengths in baboons, rhesus monkeys and cats, employing only the external respiratory nerve of Bell. We used "Bell's" nerve because:

(*a*) It was intriguing to restore the lost function of the face from "Bell's palsy" by the use of "Bell's" external respiratory nerve.

(*b*) It was exactly the size required, and was easily dissected and furnished any length desired.

(*c*) The resulting paralysis of the serratus magnus was only temporary, as the reunion by suture of the ends of the shortened nerve of Bell always brought about the restoration of the function. This was demonstrated in many final experiments.[12]

In actual experience with human beings, however, as I have detailed in the report of these cases, the sixth, seventh or eighth intercostal nerve has been employed except in the first two cases for the following reasons:

(*a*) The intercostal nerve is more easily found than the nerve of Bell in all cases, and especially in those in which there is a thick layer of adipose tissue. Although the danger of puncturing the pleura, from the fact that the nerve is in such close proximity, must constantly be borne in mind, sufficient experience with cadavers should make one so familiar with the dissection of the nerve, as to render such a mishap out of the question. (Fortunately, one cadaver will furnish a large experience.)

(*b*) The length of destroyed facial nerve, in so many cases, necessitates the use of grafts much longer than we had anticipated. In such cases, the resulting wide gap always makes the reunion of the ends of the divided nerve of Bell difficult, often impossible. The complete paralysis of the serratus magnus on one side, in such an event, is a much more serious matter than the slight paralysis and sensory impairment resulting from the loss of one intercostal nerve. It is quite unnecessary—whether the graft is short or long—to repair the divided intercostal nerve. In a long operation this saving of time is quite worth while.

(*c*) The intercostal nerves supply any desired length of graft. In the cases here reported, two required double grafts of 27 mm. and 15 mm., respectively. The intercostal nerve in these cases appeared to be decidedly smaller than the facial (in most cases it had appeared quite as large as the facial, and one graft had seemed all that was desirable). Recalling that in one of the rhesus monkeys [13] two strands of the intercostohumeral and one strand of "Bell's" nerve had been employed with a perfect recovery, in these two cases I used two strands of intercostal nerve with success.

It has been demonstrated that the employment of the smaller (finer) nerves effect more rapid and complete recovery than the larger nerves. This is due to the fact that when nerves of large caliber are employed, there may be an actual necrosis of the central axons while the peripheral axons go through a process of wallerian degeneration and regeneration only. As a consequence, the center of the nerve does not furnish the easy transit for neurons that is offered by the periphery.

12. Ballance and Duel (footnote 2, p. 68).

13. Ballance and Duel (footnote 2, p. 56).

In the finer nerves, owing to the easier nourishment of the whole caliber from the fluids in which it is bathed, until a proper circulation is established, there is less probability of a central necrosis. All the axons, both central and peripheral, will instead simply go through a wallerian degeneration and regeneration.

Thus, it is quite probable that two or three fine grafts together, while theoretically only furnishing the same diameter as one large one, may actually permit a larger number of active neurons to pass through to the distal segment, and finally make terminal connections with end-plates in the muscle fibers. Therefore, if one finds the intercostal nerve comparatively small, two, or even three, strands may be used with the assurance that they are likely to prove quite as efficient as one large one. It is probable that eventually some other nerve may be used, possibly the internal cutaneous nerve in the forearm or the external branch of the lateral femoral cutaneous nerve in the thigh.

8. Finally, I must impress on all who undertake this fascinating work, that the most careful dissection, the most successful placement of a perfect graft, may easily fail for want of meticulous care in the after-treatment. I attribute our uniform success in the matter of living grafts to the conscientious and skilful daily dressings which were carried on by my assistant, Dr. Thomas G. Tickle. The patience which must be exercised in this work is not unlike the experience of the horticulturist. A fruit-tree is planted, or a graft is made. Then comes a period of a year or more of faithful, watchful care. Neglect, for even a brief period, may result in a complete failure. On the other hand, careful attention to every detail may eventually be rewarded by an abundance of beautiful fruit or some rare flower. So with a facial nerve graft; the repair may be made with great skill; neglect in the after-treatment may easily wreck the expected recovery.

These operations may often be done in the midst of suppuration. As Sir Charles has said, "When shall we operate?" "NOW!" As my experience grows, I more and more realize the vital importance of this advice. Delay may make all the difference between success and failure.

Yet, operating in a suppurating field demands great subsequent care to prevent necrosis of the graft until it is protected by healthy granulations. In the order of their importance, the dangers that menace the life of a nerve graft, I believe, are: (1) the use of any antiseptic; (2) bungling the dressing; disturbance of the graft, by ever so slight a manipulation, before it is protected by a bed of healthy granulations; (3) pus, and (4) blood clot.

The nerve should be decompressed a good distance (from 5 to 10 mm.) above and below the graft. The graft should be laid in position with great care that no bleeding shall prevent perfect apposition of its ends with the severed ends of the proximal and distal segments. A thin

layer of dentist's gold should be laid over the whole length of the graft and decompressed facial segments. The wound should be left wide open, and lightly dressed with small bits of sterile packing gauze, wrung out of sterile physiologic solution of sodium chloride; nothing else!

This dressing should be changed daily, with such gentleness as to avoid disturbing the protecting gold. Sterile rubber tissue, perforated to insure drainage, should be laid over the raw surfaces of the open wound so that the removal of the gauze may be accomplished without causing hemorrhage. Bleeding, sufficient to necessitate wiping and removal of clots, may be disastrous from resulting disturbance of the graft.

Curiosity as to what is happening to the graft should be curbed to the point of refraining from lifting the gold foil for several days. The gold may then be gently lifted at the end or side just sufficiently to get a glimpse of the graft. If the frequent dressings have kept it free from pus, it should be found pink and healthy. If so, be content to keep on dressing daily for two weeks without another look, when, on lifting the gold foil, the nerve should be found embedded in a velvety layer of healthy granulations. Any time, from then up to a month, when the whole wound looks healthy enough to warrant it, a plastic operation to close the wound may be done.

Daily gentle massage of the facial muscles and galvanic massage, once or twice weekly, should be kept up until the muscles begin to respond to faradic stimulation.

ABSTRACT OF DISCUSSION

DR. CHARLES H. FRAZIER, Philadelphia: There is no doubt that this contribution of Dr. Duel and Sir Charles Ballance represents a distinct advance in the treatment of facial paralysis. Their operations on man and on experimental animals represent a definite step forward in the surgical treatment of this lesion. The repair of damaged nerves is not entirely unfamiliar to me. During the reconstruction period of the war, as consultant in neurosurgery to the surgeon general, I had general supervision over the management of 3,500 injuries of the peripheral and cranial nerves. In addition to these, I have in my personal files the records of 87 cases of facial paralysis.

There are two phases of this subject which suggest themselves for discussion: (1) the indications for operation and (2) the time and technic of operation.

First, as to gunshot wounds, I believe that it is proper to wait a reasonable time for spontaneous recovery of function. One can never tell whether the paralysis is due to hemorrhage, contusion or actual solution of continuity. It was my practice in military service to wait at least six months before permitting operation, and during this period there were a number of spontaneous recoveries. This was an arbitrary rule, but not an unreasonable one. I recall in one instance that the first signs of recovery were not observed until the day set for the operation, on the first day of the seventh month. According to Foerster, of Breslau, regeneration of nerves may be slow, and perhaps three or four years may elapse before regeneration is complete.

So, too, with facial paralysis complicating fracture of the base of the skull. In my experience, a large majority of patients with this condition recover without operation. As a matter of fact, I do not recall a single case in this category in which recovery, partial or complete, did not follow without surgical intervention. However, while one is waiting for spontaneous recovery, muscle tone should be preserved as a general principle by the use of galvanic stimulation, and an appropriate device should be worn to prevent overstretching of the muscles.

In the facial paralysis following mastoid operations, there is no reason for delay should the paralysis immediately follow the mastoidectomy.

How late may one operate with any hope of success in a case of facial paralysis? Twenty-two years is the longest period on record. When asked yesterday by a colleague whether it was proper to advise operation in a paralysis of ten years' duration, I said, yes, providing the muscles still respond to electrical stimulation. If they did not, one would have to assume that the muscles had been completely fibrosed.

As to the technic, the principle features of this interesting contribution by Duel and Ballance is their substitution of the nerve graft for the various anastomoses. Incidentally, I might refer to the operation successfully performed by Bunnel of San Francisco. He chiseled away the vaginal process and was thus able to shorten the course of the facial nerve by 8 mm. and effect an end-to-end anastomosis. Whenever the defect is no greater, this technic may be given preference.

As for the use of the graft in the repair of defects, may I remind you of the work of Huber under the auspices of the surgeon general's office in 1918 and 1919. Huber demonstrated the feasibility of using a nerve graft, singly or in cables, of three or four units, to repair defects in the peripheral nerves. Not only was he successful in the use of the fresh homograft, but in grafts that had been in storage for a period of eighty days.

My experience with the surgical treatment of facial paralysis has been limited wholly to nerve anastomoses. I have used the spinal accessory and the hypoglossal nerves and the descendens hypoglossi, which, I believe, is the nerve of choice. Obviously the advantages of the nerve graft, as advocated by Duel and Ballance, over nerve anastomosis is in the restoration of expressional movements. It has been known for a long time that by anastomosis of the facial nerve to any one of the three mentioned nerves, one may reasonably expect the restoration of facial symmetry in repose, the restoration of certain isolated movements such as closure of the eyelids or movements of the angle of the mouth, but nothing more.

To be able to restore expressional movements, as smiling, laughing and crying, and Duel and Ballance say that this unquestionably can be done with the nerve graft, must be recognized as a distinct step forward in the surgical treatment of facial paralysis.

DR. ARTHUR B. DUEL: Much of the discussion has hinged on the subject of various anastomoses and resulting associated movements, a question which I shall ask Sir Charles to answer from his long experience. With regard to the inquiry about the viability of the nerve, I have always employed living autoplastic nerve grafts immediately after removal. As to the plan of suturing the proximal segment of the nerve removed from its own canal—thus gaining 8 mm. by cutting across the triangle—to the distal segment, stretched up to it, as suggested by Mr. Martin, I think that procedure, though very ingenious, is unnecessary and quite likely to be followed by less success than the simpler one of allowing the nerve to remain in its bed and bridging the gap, no matter how long, by an autoplastic

graft. I believe that the trauma resulting from manipulation of the proximal and distal segments is apt to vitiate the result.

There is one problem brought up in this discussion which might be theoretically dealt with at length, but I believe it is better to continue experimenting with animals before trying to settle all these points in theory. I refer to the question of operating for Bell's palsy by decompression. I have many experiments in mind which, when completed, may make opinions more convincing.

Some one said he did not agree with the idea of immediate exploration and repair in cases of facial palsy directly following injury at the time of mastoid operation. I must reiterate my recommendation very earnestly. I should like to ask what advantage there could possibly be in waiting in a case in which one did not know (and who does know?) how much injury had been done. It is much easier to explore and determine immediately the nature and extent of the injury than to be harassed by uncertainty. The great advantage is that if one finds a definite injury in which a portion of the nerve has been destroyed, the result of placing a graft in the gap from end-to-end is sure to be more perfect than if one waited six months, or a year, lured on by a partial restoration of function, or, what is much more probable, in the end chagrined by complete facial palsy. Decision and prompt action in the latter case would have averted a catastrophe. If, on the other hand, exploration had revealed that there was no real destruction of nerve, the operation would not have done any harm. The surgeon would only have safeguarded his patient and reassured himself. In other words, if exploration had disclosed a compression by fracture of the canal, he would have lifted the compressing bone away; if it had disclosed a slight injury, decompression above and below would have insured recovery; but, if it had disclosed a badly wounded nerve with loss of substance, a properly placed autoplastic graft, at that time, would have insured restoration of function in place of a certain complete palsy. In each instance the patient would have "had a better deal," and the surgeon should have slept better nights.

I should like to ask Sir Charles to speak on the questions raised about the various forms of anastomoses.

SIR CHARLES BALLANCE, London, England: I will illustrate my remarks with slides.

I. Section of a graft in a region where 2 mm. of facial nerve were removed from the fallopian aqueduct and replaced by 2 mm. of the nerve of Bell. A junction of the proximal central portion of the facial nerve with the graft resulted after five months.

II. Graft region in a cat, 2 mm. of Bell's nerve, replacing 2 mm. of the facial nerve in the fallopian canal. New axis-cylinders replace most of the old graft after six months.

III. Middle of a graft in a rhesus monkey, 7 mm. of Bell's nerve and 2.7 mm. from the intercostohumeral nerve being used. These grafts were completely successful. The animal lived nine months.

IV. Section of geniculate ganglion. The central portion of the facial nerve was dissected out. We found that the central portion showed geniculate ganglion cells.

V. Section of the middle of a graft from a baboon which lived more than twelve months. Twelve millimeters of facial nerve was reversed in the fallopian canal. The nerve fits accurately in the aqueduct so that no sutures are required. If they are necessary, we use iodized silk.

VI. The risorius muscle. If we stimulate the muscles through the skin, contraction of the risorius occurs. Some times the other facial muscles contract when we stimulate the facial area of the opposite cortex, but the risorius fails to contract. I have asked physiologists about this, but have not been able to get information. We are as ignorant as when we sought advice.

VII. Risorius of the gibbon. Last Saturday we spent making a final examination of a baboon, Maggie, in which anastomosis of the glossopharyngeal and facial nerves had been done nearly two years before. Under anesthesia, in this baboon, there was rhythmical spontaneous spasm, contraction of the risorius and of the muscles of the upper and lower lip. The risorius pulls the mouth backward. This spasm is sometimes seen in patients with partial recovery from facial palsy. It is called the winking spasm and is produced by touching the cornea, but it occurs without winking also. Later in the examination, the cerebral hemispheres were removed. The spasm was more marked and the contraction excursions were wider. Previously, on stimulating the opposite rolandic facial cortex, no response was obtained in the right facial muscles.

We have had many experiments in which the glossopharyngeal nerve was united with the facial nerve. By stimulating the opposite rolandic cortex, we got a definite contraction of the muscles of the right side of the face. The facial area of the rolandic cortex in the baboon gave no response. We are always encountering something we have not seen before. On removal of the cerebrum, the spasm was greatly accentuated. We believe the recovery of the facial muscles was a recovery from the striate level and had nothing to do with the cerebral cortex.

In many cases we have used the nerve of Bell. This slide shows a microscopic section of the nerve stained by the Strobe method. The axis-cylinders stain blue.

Dr. Duel has spoken of the intercostal nerve. But we have simplified the operation by taking a nerve from an easier place, e. g., the internal cutaneous nerve. It does not matter whether a motor or a sensory nerve is used, the result is the same. In one baboon we took 10 mm. from the nerve of Bell in the axilla and replaced it by 10 mm. from the internal cutaneous nerve from in front of the elbow. The result was that we obtained contractions of all the serrations of the serratus magnus.

Experiments on the cortex of the baboon: We found a little vessel almost always present draining the sulcus at the base. Above this vessel the movements of the thumb and movements of the fingers are represented. Below the vessel are represented the movements of the muscles of the opposite side of the face. We always looked for that little vessel to show where the muscles of the face are represented. Below the facial area, stimulation gives contractions of the muscles of the opposite side of the tongue. Sometimes the nerve fibers of the tongue and of the face run along together.

In a section of the baboon's skull from the Hunterian Museum, I have marked various nerves—trigeminal, facial, glossopharyngeal, hypoglossal and fifth nerves. When the brain is removed, the facial nerves and the other nerves appropriate to the experiment are stimulated.

After an experiment on anastomosis, the facial nerve is removed or pulled out of the aqueduct of Fallopius. Sometimes 3 cm. of facial nerve is avulsed by slow gentle traction; in fact, it is pulled out of its attachment to the side of the brain at the border of the pons. A river of cerebrospinal fluid escapes from the aqueduct. No response on stimulating the facial nerve at the internal auditory meatus on that side of the face ever followed. We blocked the aqueduct with gold leaf. If there was ever any chance at all of the union of the two ends of

the nerve, the experiments would be vitiated. When we have done a graft of the facial nerve, we obtain contraction of all the muscles of the face on that side, equal to that on the side on which operation has not been performed. In the baboon, Maggie, we stimulated the facial nerve and no movement occurred; then we stimulated the glossopharyngeal nerve and obtained contraction of all the muscles of the face on that side. That is a wonderful thing. It is obvious that association of the various cranial nerve centers in the medulla oblongata is such that the impulse has jumped from the facial to the glossopharyngeal nucleus. In other cases we have dissected the neck up to the base of the skull, exposed the nerves and then obtained contraction of the muscles of the face.

Vagus nerve: We have performed anastomosis of the superior laryngeal facial nerve; later we have stimulated the vagus nerve at the posterior fossa, and have obtained contractions of the muscles of the face. This research is just commencing. It will take years to draw conclusions. We have united the auriculotemporal nerve to the facial nerve and complete recovery of the use of the face results; later, when the brain was removed and the facial nerve stimulated, no response occurred, but when the fifth nerve was stimulated, all the muscles of the face contracted.

Diagram of the cranial nerve centers in the brain stem: The facial nerve impulse goes down the pyramidal tract from the cortex and is arrested by the removal of the facial nerve in the aqueduct. In the auriculotemporal nerve the nervous impulse jumps from the facial nerve of the anastomosis in the nucleus to the nucleus of the fifth nerve. All the cells in the brain stem have at least fourteen connections with other cells. Human brains have a network of fibers like a telephone exchange, only the connecting fibers are innumerable. The call of the motor end-organ in the muscle, we think, explains the jumping of the nerve impulse from one cranial nerve center to another in the medulla.

When a surgeon asked if the graft was viable, I should say that there is nothing unique about a nerve graft. It acts like a bone graft. Some cells live and some die, but the life of a bone graft can be followed by roentgen study and that cannot be done with a nerve graft. It is alive in the body and is comparable with a bone graft. I was delighted to meet Dr. Frazier here. I have known him for thirty years, and have great admiration for his work. One man spoke of doing an anastomosis of the spinal accessory nerve. I did one in 1895. I decided to unite the facial with the spinal accessory nerve. I followed this procedure in many cases. I think it was a mistake. It caused dropping of the shoulder and deformity of the neck. I gave up using anastomosis of the spinal accessory nerve and also of the descendens noni. One does not set emotional expression with these anastomoses. If one reads a paper on a subject, the medical profession is under the impression that one's knowledge is limited to this subject, and all cases of this type are referred to one. I found the hypoglossal nerve was most satisfactory for giving emotional expression, but associated movements are obtained during the act of swallowing. In 1920, I performed an anastomosis of the hypoglossal nerve on a beautiful woman, and three years after I happened to be invited to a dinner at her house. As I was not famous enough for the place of honor, I sat opposite her, and on each side of me sat two beautiful young women. It was impossible not to watch the face of my former patient, and one of the young women remarked to me, "Oh Sir Charles, is that the result of your great triumph in surgery." In talking to my patient there was no contraction of the face, but in swallowing there was marked ripple of muscular contraction which spread over the face. This is a very serious disadvantage. The result is plainly shown in operations on the baboon. I performed anastomosis of the descendens

noni on a rhesus monkey; when I gave that monkey a banana, as he swallowed, the lip went up and the pinna twitched as the eyelids closed. This is true in monkeys and in man. Surgeons should sit opposite their patients at dinner and see their results. Dr. Frazier says all of his patients with fracture of the base of the skull recovered from facial paralysis. Not all persons recover. Some with serious cases have permanent facial palsy. They may recover their hearing. This would be a difficult class with which to deal. As to operating after many months, I have had patients come to me from all over the world, with facial paralysis of two or three or more years' standing. Often there is but partial recovery. These persons with old cases often have spasmodic tic as well. There is no special time for operation, but early operation avoids the disaster of delay. Relief of compression in Bell's palsy cures the palsy, and delay in operation is unjustifiable.

CONGENITAL ATRESIA OF THE ANTERIOR NARES

REPORT OF TWO CASES IN SISTERS

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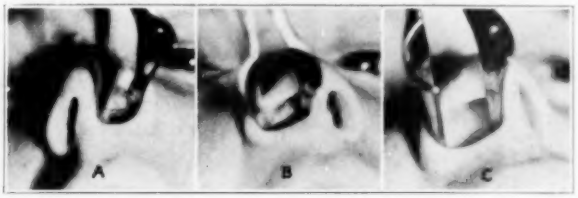
Congenital occulsion of the anterior nares is met with rarely. A survey of the literature has failed to reveal more than a half dozen cases.[1] According to Thomson,[2] this condition is caused by a "web of skin at the junction of the vestibule with the nasal chamber proper; it may be partial, complete, unilateral or bilateral."

The etiology is not clearly understood. Cheatle [1b] suggested that the closure may be due to an inflammatory process during intra-uterine life.



Photographs showing (*A*) small opening in the left nares and (*B*) constriction of the right nares in case 1 and (*C*) complete closure of the right nares in case 2.

Williams [1a] mentioned syphilis and trauma. The cases here reported occurred in two sisters with congenital syphilis. The essential findings are briefly summarized as follows.

REPORT OF CASES

CASE 1.—Bessie W., aged 18, was admitted to the hospital for treatment for congenital syphilis. The Wassermann test of the blood was 4 plus, and that of the spinal fluid was negative. Physical examination revealed an undernourished, underdeveloped white girl showing Hutchinson's teeth, saddle nose, cervical adenopathy and cryptic tonsils. The heart and lungs were normal. The blood pressure was 110 systolic and 70 diastolic. There was bilateral hallux valgus. Neurologic examination gave negative results. The pupils reacted promptly to

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1. (*a*) Williams, P. W.: Proc. Laryng. Soc., London **4**:32 (Dec.) 1896. (*b*) Cheatle, A. H.: ibid. **9**:98 (April) 1902. (*c*) Grimmer, G. H.: ibid. **13**:54 (April) 1908.

2. Thomson, St. Clair: Diseases of the Nose and Throat, New York, Cassell & Co., 1926, p. 127.

light and in accommodation, and ophthalmoscopic examination gave essentially normal results. Examination of the nares showed marked constriction in the anterior chamber of the right side and only a small opening in the left nares, just sufficient to admit the tip of a probe. The patient was able to inspire with difficulty through both sides, but expiration on the right side was impossible.

CASE 2.—Gertrude W., aged 16, was also underdeveloped and undernourished and of the same adenoid type as the patient in case 1. The stigmas present consisted of saddle nose, mouth striae and an external squint on the left with limitation of the left internal rectus. The teeth were irregular, but not of the Hutchinson variety. The chest was normal. There was a mitral systolic murmur; the blood pressure was 115 systolic and 80 diastolic. Pes cavus was noted. Neurologic examination gave negative results. The pupils reacted to light and in accommodation. Ophthalmoscopic examination showed structural blurring; the Wassermann test of the blood was 4 plus, and that of the spinal fluid was negative. Examination of the right anterior nares showed complete closure. The tip of a very small probe could be inserted in the constricted opening of the left side. Inspiration was possible on this side, but there was no expiration. In both cases the atresia appeared to consist of a web of thin cartilaginous material. The condition had been present in both sisters since birth. Unsuccessful attempts had been previously made to remove the constriction, but following operation the surgical openings had reclosed.

SUMMARY

Because of their rarity, two cases of congenital anterior nasal atresia are presented. As far as can be ascertained, these are the first cases reported in sisters with congenital syphilis.

LARYNGOSTROBOSCOPY IN THE PRACTICE OF OTOLARYNGOLOGY

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PHYSICAL BASIS OF STROBOSCOPY

Stroboscopy is based on the following facts of physiologic optics: A light impression impinging on the retina will be retained for a longer time than the time of presentation of the object whence that light impression comes; that is, the effect of retinal stimulation will continue after the physical stimulation proper has ceased. This effect is called the positive after-image. The interval between the successive retinal stimulations is such that each stimulation merges with the still present effect of the preceding one. The minimal interval at which this occurs has been variously estimated as from one fifth to one seventh of a second; that is, when two stimulations follow each other at an interval of less than from one fifth to one seventh of a second, separate stimulations recognizable as distinct from each other are no longer sensed.

The observation of a luminous body through the equidistant fenestrations in a rotating opaque disk,[1] the speed of rotation being regular and constant, gives the impression of a continuous stream of light in spite of the interruptions of the light by the opaque portions of the disk, provided the time interval of the light interruptions is no longer than one fifth of a second. The interruptions of the beam of light are not perceived. There is merely a diminution in the intensity of the beam because of the decrease in the quantum of light falling on the retina as the result of the interruptions.

On observing an object undergoing harmonic motion or regular periodic movement, such as a tuning fork executes, for example, by means of such a rotating disk, the object will appear to be stationary if the period of interruption one's vision of it receives and the period of its own movement are identical in duration.

By way of illustration, the periodic motion of an object under observation is represented by a zigzag line, and the period of its illumination, by equidistant quadrangles, which correspond to the fenestrations in the stroboscopic disk (fig. 1).

Since the interruption of illumination and the period of motion are equal, the impressions falling on the retina will come only from point *A*, and, to be sure, always in the same identical plane. Only that point in the phase of motion which is illuminated will be perceived, and since it is the same point in the period which is always illuminated in such a case, the impression will be a stationary one, and not one of movement.

1. Such a disk is called a stroboscopic disk.

The point from which this light impression comes, indeed, will change if the period of interruption of illumination has a temporal value different from that of the period of motion under observation, that is, when it is longer or shorter than the latter.

For an object with a periodic to and fro motion, the motion is again represented by a zigzag line (fig. 2). Further, the period of light interruption is considered to be one-tenth longer than the time of one complete vibration of the object under observation. The eye, then, will see the second period of vibration at a point where it is after having already executed one tenth of its course, the third period at a point where it is after having gone through two tenths of its course, and so on up to the tenth tenth of its period, one complete vibration frequency

Fig. 1.—Periodic motion of an object, represented by the zigzag line, and period of its illumination, represented by the equidistant quadrangles.

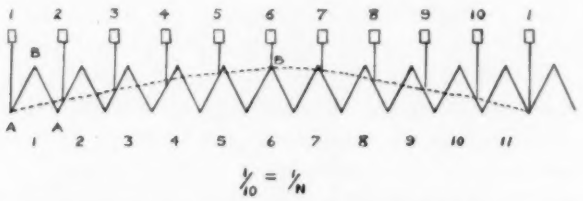

Fig. 2.—Periodic motion of an object with to and fro movement. *A-B* indicates to movement; *B-A*, fro movement, the two phases in one complete double vibration. *N* indicates vibration frequency of the object, 10 double vibrations per second. The quadrangles indicate periods of illumination of the vibrating object. *1/N* indicates the difference in time between the periods of the vibrating body and the periods of interruption of illumination.

having been executed, the beginning of the eleventh illumination coinciding with this end-point. When these points are fused, they give the impression of continuous motion, of one prolonged vibration, even though the object under observation makes eleven individual vibrations. In other words, the stroboscopic analysis of the period has made the vibration appear as though it were eleven times as slow.

Generalizing this mathematical fact by representing the discrepancy as posited by the temporal difference (which involves the factor of speed) between the period of motion of the vibrating body and the period of interruption in its illumination by the algebraic symbol 1/n, the impression of one single vibration will have been gained in the time

the vibrating body has made n + 1 vibrations. The less this discrepancy in speed, or stating it another way, the greater the value of n, the more vibrations must actually be executed in order to gain the impression of one complete vibration. When this temporal difference does not exist, that is, when the period of light interruption and the period of vibration are equal, the vibrating object will appear at rest, and, indeed, at rest at that point in its phase of motion where it is caught by the illumination.

APPLICATION OF STROBOSCOPY TO LARYNGOSCOPY

This principle of physiologic optics was discovered by two men at the same time, in 1832 (Stampfer of Vienna and Plateau of Brussels). They applied it to the study of experimental physics, for the purpose of investigating the vibrations of strings and reeds, and the observation of objects with high frequencies of motion. It remained for Oertel,[2] the renowned internist, to conceive of its application, in 1878, for practical purposes in the examination of vibration of the vocal cords, by combining it with laryngoscopy—hence the term laryngostroboscopy. This method consists merely of examining the larynx by means of interrupted illumination through the agency of a rotating stroboscopic disk.

The pitch of the human voice, which varies in range from about 60 to 1,500 double vibrations (DV) or more per second, depends obviously on the vibration frequency of the phonating cords. The less difference in time there is between the period of vibration of the cords and the period of their interruption to illumination, the slower can the frequency of their vibration be made to appear. For example, suppose the difference in time between the two periods referred to is one two-hundredths of a second, that is, that if during 200 vibrations of the cords their illumination is interrupted 201 times, the impression resulting would be as though the cords had made only one vibration in one second.

The human voice is posited chiefly between E and c^3. E has 81 DV, c^3 has 1,025 DV, and a^1, one of the mid-points, has 435 DV. Each vibration consists of to-and-fro elements, hence the term double vibration. Suppose one wishes to analyze the motion of an object having a vibration frequency of 81 DV per second, which is in the vicinity of the lowest tone emitted by the human voice. It is evident that 1 DV of this frequency would require the one eighty-first part of a second, which is already about sixteen times less than the time interval necessary for the differentiation of two successive impressions. In tones representing higher frequencies, this time interval is much smaller. The greater the frequency becomes, the smaller does this interval grow. The practical application of laryngostroboscopy lies in this fact.

2. Oertel: Das Laryngo-stroboskop und die laryngo-stroboskopische Untersuchung, Arch. f. Laryng. u. Rhin. **3**:1, 1895.

Returning to the aforementioned example, in which the difference between the two periods is one two-hundredth of a second, the vibration frequency being 200 DV and the interruptions of illumination 201, the single vibration which seems to occur in the period of one second as a result is really a reconstructed vibration. It is an analysis of the components of rapid periodic motion into slow motion, having exactly the same form as each of the original 200 DV. Because the eye sees the first one two-hundredth part of the vibration during the first illumination, the second one two-hundredth part during the second illumination, the third one two-hundredth part during the third illumination and so on, there is constructed in the period of one second one single vibration, the form of which can be observed directly. This is the basic principle of the slow motion film. In the case of laryngostroboscopy, the eye is the camera.

Putting this theory into practice, suppose that one desires to make adjustments so as to observe one vibration of the cords in the interval of one second. To bring this about it would be necessary for the stroboscope to maintain a speed of rotation which would cause the number of interruptions of light to be one more than the corresponding frequency of vibration represented by the tone emitted by the cords. This necessary speed of rotation can be regulated by the ear. Air blown through a tube so placed that it allows the air escaping through it to hit the fenestrations of the rotating disk will produce a tone on the principle of the siren. The principle of the siren involves the interruption of a current of air, in this case by means of the fenestrations in the disk through which the current of air finds egress. When this interruption is periodic, a tone of a definite pitch is produced, the frequency of which is identical with the number of interruptions the current of air receives. Thus, the pitch of such a tone depends on (1) the number of revolutions of the disk per second (the speed of rotation) and (2) the number of fenestrations in the disk. For example, a disk with 20 fenestrations revolving at a speed of 20 revolutions per second will produce a tone of 400 DV per second, which is in the vicinity of g^1. The resulting tone indicates, by corollary, the number of light interruptions occurring per second, viz., exactly the same number as the frequency of vibration of the tone produced. Further, given the number of fenestrations and the pitch of the tone, one can easily determine the number of revolutions made by the disk. The principle is the same whether the siren produces the tone or whether the tone is produced in the larynx, for in both cases what is involved is a comminution of the current of air.

To analyze the vibration of the cords on a given tone into compoments which will effect the impression of one vibration per second, it will be necessary, as already mentioned, to control the speed of rotation of

the disk in such a way as to cause the number of light interruptions to be one more than the tone has vibrations. The siren tone of the disk, therefore, will need to have one vibration more than that of the cords. In other words, the less the difference between the frequency of the tone of the disk and that of the cords, the slower will be the analysis of the vibration of the cords. When the two pitches are identical, the cords will appear to be stationary, appearing at rest in the position in which they happened to be caught by the illumination. For example, in a case undergoing stroboscopy, in which the tone taken has 200 DV and the disk has 20 fenestrations and makes 10 revolutions per second, either no change will occur as compared with the usual laryngoscopic examination, or the cords may be caught in their open phase, in which case they will be seen as just about to meet and apparently stationary.

The investigations of Hender and Kruenzl in connection with stroboscopy are important. They demonstrated that not even the art singer can take a tone exactly at the same pitch, in the physical sense, as the one presented by the siren. Even if the singer should happen to hit it exactly, it is impossible for him to maintain it at the same physical pitch, the tone increasing or decreasing slightly. These variations, however, are so slight that one is not capable of differentiating the discrepancies involved by means of one's hearing. The differences are 1 per cent or less, as a rule, and are rarely more than 2 per cent. This is below the differential threshold for pitch discrimination. To be sure, the fineness of such discrimination by means of hearing varies with the subject. There are ears which can perceive differences in pitch of 3 or 4 per cent, while the threshold for other ears is placed at 8 per cent. Of course, a musically trained ear is more likely to have a finer threshold than an untrained ear, but this is not always the case.

Oertel [2] experimented first with vibrating membranes, in which he claimed to have observed two distinct vibrating zones separated from each other by nodal lines. He claimed that he also observed nodal points in the vibrating cords. Koschlakoff [3] claimed to have made similar observations.[4] This claim, however, has since been shown to be erroneous. It is generally believed, at the present time, that this error rests on the fact that in the case of vibration of the vocal cord, especially in pathologic cases, but in normal cases as well, mucus can stick to

3. Koschlakoff, D.: Ueber die Schwingungstypen der Stimmbänder, Arch. f. d. ges. Physiol. **38**:428, 1885-1886.

4. Oertel saw in the chest register "the tones produced by vibrations of the cords in their entire length and breadth, while the head register tones originated through longitudinal distribution of the cords into various parts by the formation of nodal lines." Koschlakoff based register differences on the various degrees of vibrational distribution in which the cords took part, the chest tone involving the whole cordal mass, while the head tone involved the narrow marginal portions next to the glottic chink, which were separated from the peripheral portion of the cords by a nodal line, the movement of which could not be observed.

points along the margin of the cords, which occasionally gives the impression of nodal rest.[5]

PHYSICAL AND PHYSIOLOGIC FACTORS UNDERLYING THE SO-CALLED CHEST, MIDDLE AND HEAD VOICE REGISTERS AS REVEALED BY LARYNGOSTROBOSCOPY

A few comments in regard to vocal phenomena are necessary to indicate the significance of laryngostroboscopy. Formerly it was believed that during the production of voice the vocal cords moved up and down in a vertical plane, thus giving rise to tone. The present belief is that the column of air issuing through the trachea by means of intrapulmonic compression is interrupted and comminuted at the level of the cords by the periodic vibration of the latter and is in this way thrown into periodic condensations and rarefactions, emerging after modification in the resonating parts where the voice engendered receives its timbre or clang, as tone.

When chest register or chest voice is spoken of, reference is made to tones in which so-called chest resonance predominates in function. The function of chest resonance occurs by virtue of the fact that the production of such tones involves complete closure of the glottis, the cords just coming in contact with each other, causing a momentary cut-off of the issuing column of air, with the result that there is a recoil of the sound waves into the chest.

Head register or head tone, on the other hand, refers to those tones produced under conditions in which the glottis contains a fine slit of greater or lesser dimensions, the presence of which does not allow for the recoil of the sound waves into the chest, or at least not more than to a slight degree, the sound waves finding their predominant modification in the parts superposed on the larynx because of this fact.[6]

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5. This brings up the question as to where, in general, mucus accumulates in relation to the cords during phonation. Where does it stick or tarry during cordal rest? It rests where motion is reduced, whether it is in terms of frequency or in terms of amplitude, or where there is no motion at all. Motion is most likely to be reduced where there is inflammation, infection or degeneration of muscle tissue.

6. As long ago as 1835, before the advent of the laryngeal mirror, Lehfeldt made the correct assertion that the head tone involved only the merest marginal portions of the free edges of the vocal cords, while the chest tone was associated with vigorous vibrations of large amplitude. Johann Müller corroborated these assumptions by experimental investigation in the cadaveric larynx (Handbuch der Physiologie, Coblenz, J. Hölscher, 1840). Gottfried Weber compared the head tone with a flageolet tone and assumed it to be due to a nodal division of the cords, that is, an independent and partial vibration of the length of the cord, much after the manner of the vibration of a string. Johann Müller denied this as causal, but conceded a similarity of origin of the head tone so far as his observations of the vibrational distribution in the cord showed it to be limited to the marginal portions only, the whole length of the cord, however, being involved in the vibration.

It should strictly be borne in mind, however, that no tone is sung exclusively with either head or chest resonance, but that a certain mixture of the two is always present.

Another important register is the so-called middle register or *voix mixte* register of the French. By this is understood the production of tones possessing a mixture of chest and head resonance in approximately equal ratio. An ideal tone of the middle register contains about 50 per cent of chest and about 50 per cent of head resonance. Such a tone is most difficult to produce, and the ability to produce it is an excellent gage of what a singer can really do. This difficulty of production is explained by a simple consideration of the physiology underlying it. Such a tone depends chiefly on the form of the glottis. Complete closure of the glottis would produce a chest tone. A continually open glottis would produce a head tone. For an ideal middle register tone, there may be no complete closure of the glottis, nor may a constant opening be present. A mid-point between these two is necessary, and this is difficult to obtain. It is so difficult that it can justly be said that if it does not already exist as a natural ability, it is most difficult to learn.

Stroboscopic observation of the cords during the production of a chest tone reveals the cord as slightly humped. Normally, the mucus present, which is predisposed to accumulate at both commissures, is carried over laterally. A gradual decrease in the intensity of the tone will cause this humping to diminish to a point where the cords become nearly flat. The humpy contour of the cords associated with the production of chest tones is caused by the fact that in the chest register the musculus vocalis, or as it is otherwise known, the arytaenoideus internus, assumes the main burden of the work involved.

When a tone undergoes decrescendo, that is, when there is a gradual diminution in its intensity, the external tension of the cords is increased in the same degree as the intrapulmonic pressure or the force of breath is decreased. This tension mechanism is controlled by the reciprocal innervation of the internal and external tensor muscles of the cords. It occurs by virtue of the fact that the activity of the thyroarytaenoideus internus decreases in ratio to the increase of activity on the part of the cricothyroideus externus. At the same time the glottic closure recedes and the rima widens, and when the external tensors have acquired an activity over and above that of the internal tensors, the headtone or so-called falsetto effect is produced.

The characteristic of this clang modification thus lies in the behavior of the glottis, which is an expression of the manner of distribution of the muscular activity involved (two elastic bands, like all elastic bodies, decrease in dimension at right angles to the direction of the pull

exerted on them). This is an important factor in the spindle-like formation of the glottic chink.[7]

The middle register, thus, is produced by a gradual change in relationship between the factors involved in the tension of the cords.

Probing the cords while in the act of producing a falsetto tone reveals a resistance which indicates a condition of increased tension.

Jörgen Möller [8] demonstrated by x-ray the fact that the falsetto tone is associated with an approximation of the upper margin of the cricoid cartilage to the lower margin of the thyroid cartilage, over and above the degree of approximation observed in the case of a chest tone. This too is an effect of the cricothyroid muscles.

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7. Réthi, using Johann Müller's method, produced a chest tone by blowing into the cadaveric larynx. On touching a point near the free margin of the cord with a sound, a higher tone was produced—in fact, a head tone. He also obtained a head tone by placing a thin needle or a tensed thread parallel with the free margin of the cord. On sticking a needle through the cords from before backward through the thyroid cartilage into the arytenoid, parallel with the free cordal margin and in a plane with the lower and upper surfaces of the cords, a head tone was produced. Even one cord so treated gave rise to a head tone, while the other cord accommodated itself to it, if the free margins were not too far apart. Réthi also sprayed a larynx prepared after the manner of Johann Müller, on its upper surface, including the immediate parts associated with the sinus of Morgagni, with bronze dust, and observed the movements of the glistening particles microscopically during the process of phonation. The greatest up and down movement of these particles was seen in the medial margin of the vocal cord during the production of a chest tone, the excursion diminishing with the more forward and lateral placement of the particles. Complete cessation occurred in the region of the thyroid cartilage. In head tone, on the other hand, produced by the transfixed needle, he noticed the excursions to be much less, being greatest at the cordal margin, and growing rapidly less in a lateral direction, petering out entirely along a line almost parallel to those margins. Since he could no longer perceive any motion beyond this line, he regarded its interpretation as nodal to be erroneous, contending that the nodal lines claimed to have been seen by Oertel and Koschlakoff were illusions which could be caused by the fact that the free margin of the cord is seen to vibrate up and down, causing an angulation as the result of the limitation of its lateral distribution. This angle courses a short distance laterally, gradually disappearing, the formation of a second angle coming under way before the first has run its course. These lines should not be considered as nodal lines, because they do not differentiate diametric movement. In the middle register, according to Réthi, a wider zone vibrates than in the case of head tones, the cords being flat and the waves coursing from the free margin laterally as in head register, while in chest register the cords vibrate throughout their entire mass. A cylindric protuberance more or less parallel with the free margin can be recognized, but no wave motions as in the registers representing middle and chest tones are observed. (Réthi, L.: Experimentelle Untersuchungen über den Schwingungstypus und den Mechanismus der Stimmbänder bei der Falsettstimme, Wien. klin. Rundschau **11**:71 and 88, 1897; Die Stimmbändspannung, experrimentell geprüft, Sitzungsb. d. k. Akad. d. Wissensch. Math.-naturw. Cl. **106**:244, 1897.)

8. Möller, Jörgen: Ueber die Wirkung der Musculi crico-thyroideus und thyreo-arytaenoideus internus, Arch. f. Laryng. u. Rhin. **15**:72, 1903.

Musehold[9] has repeatedly attempted to fix the phase of closure of the cords, as well as the phase in which the cords are widest apart during phonation, by means of the stroboscope. He found it relatively much easier to fix the phase of closure than to fix the phase at which the cords are widest apart. This led him to conclude, quite logically, that the phase of glottic closure is longer in point of time than is its open phase.

A pure falsetto tone is shown by stroboscopy to be associated with the vibration of the merest edge of the inner margins of the cords. The usual falsetto tone shows a slightly wider zone of vibration.

Various investigators, notably Oertel and Réthi, claimed that the cords vibrate up and down. This, as previously stated, has since been shown to be incorrect. Vibration of the vocal cords is made up of a compound motion, the course of the excursion being upward and outward with a predominance of the lateral component.

The previously mentioned fact that the phase of closure of the glottis is of longer duration than its open phase is the simplest explanation for the recoil of the sound waves. The breath below the glottis undergoes a definite compression at the time of glottic closure, with the result that there is a rebound (the principle of force and counter-force). This represents a to-and-fro element below the glottis proper. These vibrations are especially concerned in loudly sung tones of low pitch, and hence their name, chest tones. The sudden closure of the glottis explains why the rebound occurs, the reason being simply that the issuing column of breath receives the impact of a momentary obstacle to its forward course.

In falsetto tone, this rebound is naturally wanting, because of the constant opening in the rima, the breath receiving no impact as it does against a completely closed glottis in the case of low tones, and thus its propagation has less with which to cope. The better the forward coursing occurs, the more optimally is the available resonance utilized and so much the better will the production of the head tone be. The matter of resonance also involves the necessity of the absence of pressure of the epiglottis on the larynx and the absence of factors obstructing the vibrations as they leave the larynx, such as a thick lingual radix, large lingual tonsils and markedly thickened lateral pharyngeal bands.

High and low falsetto tones must also be differentiated. High falsetto tones can be sustained for a longer period than low ones. The reason for this is that the high falsetto tone is associated with a short and narrow rima, which allows only a slight amount of air to escape. Low falsetto tones, as a rule, cannot be sustained for as long a time because the rima is wider and longer, and a sort of physiologic dissipa-

9. Musehold, A.: Stroboskopische und photographische Studien über die Stellung der Stimmlippen von Brust- und Falsett-Register, Arch. f. Laryng. u. Rhin. **7**:1, 1897.

tion of air occurs. These facts also explain why the production of *voix mixte* is so difficult, viz.: (1) physiologic dissipation of breath, since in *voix mixte* there is a fair admixture of falsetto (head) tone, and (2) the decreasing gradation of function of the external cricothyroid muscles as opposed to their antagonists, the musculi vocali (interni), for, as the activity of one increases, the activity of the other must decrease (reciprocal innervation).

As the scale is ascended from low tones in falsetto voice, the glottis undergoes a change in form, the important fact being that the glottic chink becomes shorter as the ascension continues, being finally limited approximately to the anterior halves of the cords, while the posterior halves of the cords are in close contact with each other, being pressed together, as it were, and taking no part in the vibration.

The implication, thus, is a partial glottic closure in a vocal plane representing the high falsetto tones. This assumption is based on the following facts: 1. In high falsetto tones the musculus arytaenoideus transversus and the musculus arytaenoideus lateralis exert quite a bit of pressure on the arytenoid cartilages. 2. It has been found in high falsetto tones that though the activity of the musculus vocalis (the musculus thyro-arytaenoideus internus) is involved, it is involved, however, only in its posterior portion, that portion in relationship to the posterior half of the glottis. The oblique course of the fibers of the musculus vocalis, especially in its posterior portion, is a possible, indeed a plausible, explanation for this partial innervation. It should also be remembered that the timbre of falsetto voice is conditioned by the fact that the glottis itself, because of the great external tension which stretches the cords, remains open. In falsetto voice the cords are narrower and the glottic chink is wider. The margins of the cords never make a contact, as they do in the case of chest tones.

STROBOSCOPIC OBSERVATION OF ABNORMAL GLOTTIC BEHAVIOR

All that has been said about the form of the glottis and the mechanism of vibration of the vocal cords is important in the physiologically correct production of tone. Deviations from it will occasionally occur, even in singers who sing correctly. Generally, though, it is as the the description represents it. If, for instance, on examining a singer I should find that the glottis is closed during a falsetto tone, that fact, excluding the possibility of exception, justifies the assumption that the singer in question is singing under impulses of hyperinnervation, a condition which Flatau refers to as hyperkinesia. Such a singer already closes the glottis in the use of the falsetto register, which is the exact opposite of what one expects to find normally. What should one expect to find, then, during the production of a chest tone, where glottic closure is normally present? Not only would one be justified in assum-

ing that the production of a chest tone by such a singer would show the equivalent of a hermetically sealed glottis, so to speak, but one would expect to find nothing short of strangulation. That this is so requires an acquaintance with only a single case of hyperkinesia at a fairly well developed stage. In vocal practice this is seen continually, varying from slight degrees of constriction to an utter strangulation, which not only is reflected in the voice, but in time causes severe injury to the vocal organ. The peculiar character of the voice produced under such conditions is referred to in ascending degree as throaty, constricted, choked, strangulated. On the other hand, singers with this disturbance in coordination, for that is what the condition really is, may show an open glottic chink during the production of chest tones, which is just the opposite to what should occur normally. The presence of a closed glottis in falsetto register and an open one in chest register represents a physiologic inversion of function and signifies a more or less advanced stage of the hyperkinetic process. The meaning of this is simple. Constant hyperinnervation, effecting itself in constriction and excess tension of the parts involved, over a long period is still evidenced in the falsetto register, while in the chest register the muscles involved have become so fatigued because of years of overexertion that they are insufficient to carry out their proper functioning and lay down on the job, so to speak. This is the stage of so-called hypokinesia,[10] in contradistinction to the one of hyperkinesia referred to. At this stage a person cannot effect a prompt glottic closure in the chest register even with the utmost effort. This is often the basis of a breathy tone and the inability to initiate or sustain a *pianissimo*. It is obvious how vicious a circle this process becomes, and not always do measures for its correction succeed once it is well on its way.

The stroboscopic finding of a glottic closure in the falsetto register and a glottic opening in the chest register, an inversion of normal function, is an unfavorable sign prognostically. It has about the same significance for voice as arteriosclerotic signs have for the body. It is an indication that the organ has or is about to run out its course. Such a condition can never be completely cured, but much will already have been accomplished if by proper management and, above all, by the reestablishment of even a degree of correct function partial remission is incurred. With the proper care such improvement may last for years, a fair *status quo* being maintained. When therapy fails to be effective, the prognosis naturally is bad. Experience has taught that not infrequently a rapid ruination of the voice occurs in spite of all therapy. Here again the individual equation is involved.

On the other hand, it is important to bear in mind that in laryngoscopy as it is ordinarily carried out, the examination is usually made

10. This condition is often diagnosed as paralysis of the internus muscle.

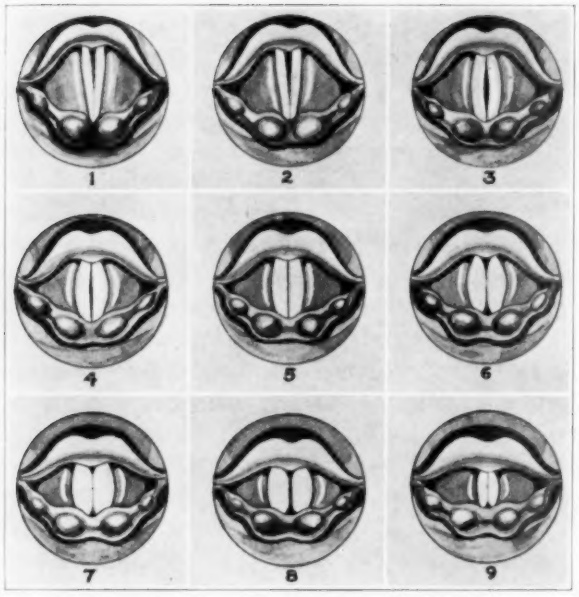

Fig. 3.—As indicated in the text, the mechanism of the different vocal registers is associated with various activities of the cords which determine the forms of the glottis. Laryngostroboscopy facilitates the differentiation of these forms. The illustrations are semidiagrammatic representations of these forms.

Explanation of Plate

1, 2 and *3:* The shape of the cords and the form of the glottis during the production of head tones. The cords are thinner and more angular than during the production of chest tones, because the rounding caused by the active contraction of the musculus vocalis necessary for the chest tone is absent. The medial margins of the cords diverge in greater or lesser degree because of the external tension exerted on them by the cricothyroid muscles, and the insertion of the lateral margins of the cords into the walls of the larynx. Under such conditions, the cords behave like any elastic mediums subjected to longitudinal stretching when they are fixed laterally. The result is a gap in the rima, which is characteristic for the head tone, the glottis never closing completely, even during phonic vibration. The so-called amphoteric tone (a tone lying in the vocal range just between head and chest registers), when produced in the head register, shows a longer glottis and thinner cords than when it is produced in the chest register. A forward traction of the epiglottis is associated with this longitudinal stretching, which causes it to assume a more vertical position than it would otherwise, and thus opens the laryngeal aditus to a greater degree.

6, 7, 8 and *9:* The shape of the cords and the form of the glottis during the production of so-called chest tones. *7* represents the closed phase; *8*, the open phase, of the vibration of the cord. In loudly sung chest tones, the cords are slightly humped. This humping is especially pronounced in their mid-portions. It occurs as a result of the contraction of the musculi vocali, which crowd the cords toward the midline. The greater the tonal intensity (breath pressure), the more does this crowding toward the midline occur (force and counter-force) and the more completely is the glottic chink momentarily occluded before the increase of intrapulmonic density caused thereby becomes sufficient to disrupt them, with the resulting rarefaction of the breath contained within the lungs, thus giving rise to a periodic oscillation of intrapulmonic pressure, which forms the physical basis of the so-called chest tone. The glottic chink, thus, is correspondingly thinnest in its mid-portion, while its anterior and posterior limits are wider. When the breath pressure is lessened, the line representing the glottic chink becomes more definitely linear, because the cordal surfaces become flatter, owing to the corresponding let-up in tension on the part of the musculi vocali. The degree of the phase of the closure is lessened, with the resulting increase in head tone register admixture and a decrease in tonal intensity, the so-called decrescendo (*4*).

5 is a diagrammatic representation of the glottic chink and relative cordal posture during the production of tones, in which head and chest tones are mixed in approximately equal ratio, the so-called voix mixte or middle voice. In low chest tones, the entire length of the glottis, that is, both the intermembranous and intercartilaginous portions, is represented by a narrow slit (open phase). As the scale is ascended in the chest register, the intercartilaginous portion of the glottis gradually becomes narrower until it is closed off altogether, the intermembranous portion remaining open. If the chest register is not maintained and the ascension in pitch makes a transition through the middle register into the head register, a gradual shortening of the glottis from the vocal process on can be observed (*5, 4, 3, 2* and *1*); see text. In the middle register the glottis, thus shortened, retains its linear form to a greater or lesser degree (*5* and *4*), while in the head register it becomes more or less spindle-shaped, ultimately assuming a more or less triangular form. This, however, being true in the vast majority of cases, is not an invariable rule. There are persons in whom the glottis remains linear, even in the highest head tones. Although the evidence is not complete, my own observations seem to point to the fact that the spindle-shaped glottis is met with more in the so-called natural singer and less in the properly cultivated and artistically trained voice.

while the patient phonates in the falsetto register. Normally, as previously indicated, the glottis will assume a form which presents a slit of greater or lesser dimensions. On that account such a finding is often diagnosed as an internus paresis or insufficiency. If after a few days the same patient is examined and happens to take a tone in the chest register, the glottis is seen to be closed, as should normally occur, and the conclusion is immediately jumped at that a cure of the internus paresis has been effected, all of which means a mistaken diagnosis. It is therefore recommended that laryngoscopic examination be carried out in both falsetto and chest registers and that the glottic forms of each be compared. Stroboscopic examination, when correctly executed, reveals the glottic form and behavior throughout the entire range of the voice. This objective evidence is important in the evaluation of vocal function.

VALUE OF LARYNGOSTROBOSCOPY IN CERTAIN ORGANIC LESIONS OF THE LARYNX

Not only is stroboscopy of diagnostic value in matters pertaining to functional deficiencies of the voice per se, but it is also of great service when applied to the determination of the influence of organic conditions on laryngeal function. I shall consider first the general diagnostic entity of laryngitis.

Ordinary laryngoscopy shows reddening, swelling and other gross changes in the physical attributes of the cords, but what is actually happening directly in the vocal cords themselves, only laryngostroboscopy can indicate. At the present time, the vocal cords are considered as cushioned *contre coup* devices. The phrase is purely descriptive. It is logical, in that the cords are indeed cushions which strike against each other during vibration. Since Ewald [11] first presented this idea, it has come to be generally accepted. The mathematical formulation of the activity of such a vibrating mechanism has not as yet been set down. The vocal cords, therefore, have been mathematically considered as membranous reeds striking against each other. The mathematical formula which describes the vibrations of reeds is, thus, the closest mathematical description of the vibration of the cords. This formula is as follows:

$$N = \frac{L}{2L}\sqrt{\frac{g \cdot p}{qu. \; s}}$$

N = vibration frequency (pitch)
L = length of vibrating body (vocal cord)
g = a constant (acceleration of gravity)
p = tension of vibrating body (vocal cord)
qu = diameter of vibrating body (cross-section of vocal cord)
s = specific gravity of vibrating body (vocal cord)

11. Ewald, Richard: Physiologie des Kelkopfes, in von Heymann, Paul: Handbuch der Laryngologie und Rhinologie, Vienna, Alfred Hölder, 1898.

In a case of inflammation, for instance, qu will be increased, and this will involve an increase in s, since inflamed tissue has a definitely increased specific gravity over that of normal tissue. This is already the case when the tissue is permeated with serous fluid, although not to so great a degree as when the inflammation is more severe.

The element of tension, p, is secondarily involved. It is logical to suppose that a substance which is increased in diameter and which, in addition, has gained in specific gravity as the result of an inflammatory reaction will behave quite differently when undergoing tension than it would under normal conditions.

Thus, in this formula, inflammation will affect the value of qu and s, with the secondary involvement of p, leaving whatever compensation is necessary to be performed by L. However, L cannot be appreciably changed, especially not when in a condition of inflammation, and thus it is self-evident that N becomes seriously involved. This formula explains all voice disturbance in laryngitis. Thus N, the vibration frequency of the cords, is reduced; there is an impairment of phonation, and there is hoarseness, but first and foremost, the pitch of the voice is lowered.

Stroboscopically, in cases of laryngitis still in the acute stage for the greater part, one often sees differences between the two cords. For example, one cord may make a much smaller excursion than the other, or it may vibrate at a lower frequency than the other.

The fact that mucus accumulates at points on the cords where their motions are diminished or absent has already been mentioned. The significance of this is that such a finding stroboscopically is an indication that the cord is affected most severely at such points. It is interesting to follow the course of laryngitis stroboscopically. For example, in a case in which the condition as such, in a general way, after a period of two days does not show any essential change, the stroboscopic picture, as compared with the findings at the time of onset or when the process was at its height, reveals definite changes, one or both cords showing marked improvement in the amplitude of vibration. The discrepancy between the frequency of vibration of one cord and that of the other can almost be seen leveling itself out under one's very eyes. When this is the case, the patient can be told with certainty (and this is important to patients whose work depends on the use of the voice) that a definite improvement can be expected within an interval of hours. This is especially important in singing practice, and is repeatedly being corroborated by experience.

The cause of hoarseness, otherwise known as *vox anserina* or dysphonia, is attributed to the motor discrepancy, as to both form and number, between the two vocal cords. This is on the basis, in the majority of cases, to a greater or lesser degree, of catarrhal disease.

There are, of course, cases in which the culpable agent is not so much the inflammation as a change in the tension attributes of the cords, which is easily explicable on the basis of that which has already been described in relation to the various functions of the vocal cords.

What has been said about acute laryngitis is also applicable to chronic laryngitis. In the acute forms the facts described are more outstanding.

Stroboscopic examination also serves to indicate different therapeutic measures (when qu and s are interfered with), with special reference to the time for, and the type of, vocal gymnastic.

A study of the problem presented by lesions of the recurrent laryngeal nerve is substantially aided by stroboscopy. For example, in a case in which routine laryngoscopy shows an absolute fixation of one cord, the conclusion is reached that there is a complete paralysis. There are cases like this one, however, in which the cord affected is shown by stroboscopy to be capable of some movement, no matter how slight, be it in amplitude, frequency or both. In any event, some vibration is present. This justifies the conclusion that certainly not all of the nerve fibers had undergone destruction; that, perhaps, for example, as the result of pressure or cicatrix, a large number of the nerve fibers had been destroyed, but there were still some fibers which functioned and were sufficient to set the cord into ever so slight a vibration, and which, having escaped detection by the eye, succumbed to the scrutinizing analysis of stroboscopy.

On the other hand, stroboscopy can tell much in cases viewed from another angle. For instance, suppose that in a case of recurrent paralysis, no cordal movement whatever was perceived stroboscopically or laryngoscopically. Suppose also that the case was not associated with any severing of the nerve, but that the picture was due to the pressure of a cicatrix, to a tear or to an adhesion. Further, suppose that the usual therapy for recurrent paralysis was instituted, and that there was not the slightest evidence of any improvement so far as the voice was concerned, besides the evidence supplied by the laryngostroboscopic examination, the condition having been observed daily over a period of time. Then, one day, stroboscopy suddenly showed some improvement in the paralyzed cord which could never have been detected by ordinary laryngoscopy. This discovery justifies the inference that all the fibers of the nerve were paralyzed temporarily only, and that the therapy was beginning to prove effective. I recently saw such a case in which further and persistent therapy brought about a fairly good result.

Stroboscopy is also of value in comparing the plane of one cord with that of the other, or what I term the *niveau comma* of the cords. Indeed, ordinary laryngoscopy will show this if it exists, but it is much better evaluated when viewed in motion (slow motion). Where the *niveau*

comma is slight,[12] as it is, for example, in many cases of recurrent or internus paralysis, it shows up better in motion (that is, in the position of phonation) than during rest. As is well known, in the great majority of cases the affected cord is lower than the healthy one.[13] There are indeed, instances in which the lower position of one cord may be physiologic and not pathologic. Likewise, the overriding of one arytenoid cartilage can also occasionally be seen in an otherwise absolutely normal case. *Niveau* differences cause dysphonia by interfering with the periodic comminution of the air current.

Stroboscopy can tell more from the point of view of function in all forms of neoplastic development in the cordal tissue, as well as in hemorrhagic and ulcerating processes, than is possible by means of the ordinary laryngoscopic examination. The most important cases in this connection are those of singer's nodes. Stroboscopy shows clearly that often in such a case closure of the vocal cords is not at all interfered with, the node being located sometimes as much as 1 mm. from the margin of the cord, and in no way disturbs the comminution of the air current into periodic components. There are singers who have had these nodules or papillary structures for years without the development of any disturbance in the production of voice.

Stroboscopic data serve as an excellent index of the extent of effect on function and degree of recession in all of these organic conditions. There are, naturally, many other occasions for its use, as in the observation of the pareses connected with mutation, affording much information for the evaluation of clinical findings.

CONCLUSION

The foregoing discussion has indicated why stroboscopic examination of the larynx is a valuable supplement to ordinary laryngoscopy and an absolute requisite for vocal work. It enables observations of the function of the cords under both physiologic and pathologic conditions, and affords a source of valuable information concerning the diagnosis, prognosis and effects of, and indications for, treatment in some organic, as well as in all functional, disturbances of the voice.

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12. Normally, as a rule, both cords are in the same plane, the niveau comma then being zero.

13. According to Spiess, this occurs in about 90 per cent of cases.

ALLERGY AND THE CYTOLOGIC EXAMINATION OF NASAL SMEARS

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In the past few years the various manifestations of allergy have received more attention than formerly. Stimulated by the work of Duke, Cocoa, Vaughan and others, the medical profession has been more alert in the recognition of conditions of this nature and has followed cases through until the exciting agents have been identified.

Since in many cases of sensitization there are symptoms that are referable to the nose or eyes, it behooves the rhinologist to have a sufficient understanding of the nature of these cases in order that an error in diagnosis is not made when the patients present themselves.

Many patients visit the rhinologist seeking relief from such symptoms as nasal congestion, a watery nasal discharge, headache, sneezing or itching of the eyes, nose, mouth or pharynx. These symptoms should suggest the possibility of sensitization. Some patients, in addition to any of these symptoms, may complain of other conditions, such as urticaria, asthma or abdominal symptoms, which may also serve as a clew in the identification of the cause of these symptoms.

The exciting agent in an allergic case may fall into one of three classes: The first consists of the inhalants, such as the various pollens of weeds and grasses, house dust and danders. These are perhaps the most common ones causing rhinologic symptoms. The foods are the second group of allergens, and the third group embraces anything with which the patient may come in contact, such as glue, fur and rubber, which may serve as an exciting agent.

It has been noted by various workers that allergic persons are usually sensitive to several agents. Occasionally, a patient reacting to only one allergen is encountered. Seasonal variation is to be expected with the various pollens and grasses. Perennials cause symptoms which are present throughout the year, as is also the case with house dust, feathers and the common foods.

Cytologic examinations of smears taken from the nasal mucosa and from the antrums have been made by various investigators, namely,

Sewall and Hunnicutt,[1] Arbuckle[2] and Dean.[3] It is thought that the finding of polymorphonuclear leukocytes indicates an active infection, lymphocytes being found in the chronic infections and eosinophils in cases of sensitization. It is considered that the finding of polymorphonuclear cells is not of any particular significance, since they are found so frequently in persons who have a slight cold or a mild nasal congestion.

OBSERVATIONS

Our series of thirty-four cases is composed of patients who consulted a rhinologist because of symptoms referable to the nose or eyes and who from the history or examination, including the cytologic study of nasal smears, were suspected of being allergic.

The history included questioning as to the nasal congestion, its initial appearance, its duration and any factor which appeared to influence it. The character of the discharge and itching of the eyes, nose, mouth or pharynx were also inquired about. Also any other possible allergic manifestations and any family history of asthma or hay fever were noted.

The study included the usual nasal examination and transillumination of the sinuses, with the taking of a smear from the nasal mucosa for cytologic study. In the smear 100 cells were counted and the results expressed in percentages. In a few cases 100 cells could not be found. Rather late in the series, a routine differential blood count was included.

If the history was suggestive of allergy and the cytologic examination showed an eosinophilia, the patients were referred to the allergist for cutaneous tests of sensitivity. Occasionally, the presence of eosinophilia could not be demonstrated, and these patients were referred for testing, owing to the history that they gave, which suggested allergy.

The tests of the skin consisted of the usual scratch and intradermal tests. The usual number of allergens were used in the tests. The pollens and danders common to the locality, the usual foods and some contact allergens were used. All cases in which an eosinophilia was present in the nasal smear gave a positive reaction to one or more allergens. In the cases in which the patients were suspected of being allergic from the history, without eosinophilia, a positive reaction was obtained in the greater majority.

In tabular form are given the findings in nineteen of the patients included in this series, which illustrate the various factors mentioned.

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1. Sewall, E. C., and Hunnicutt, Leland: Cytologic Examination of the Antrum, Arch. Otolaryng. **10**:1 (July) 1929.

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Summary of Nineteen Cases

Case, Name, Age	Complaint	Examination	Smear	Sensitization Tests
1 Miss V. G. 22 years	Frequent head colds; profuse watery nasal discharge from nose and posteriorly into throat; wheezy respiration in damp weather; sneezing marked; no seasonal variation; no itching of eyes, nose, mouth or pharynx	Nose: polypi in each middle meatus; mucous membrane pale; septum in pressure contact with right middle turbinate; frontal and maxillary sinuses clear on transillumination; pharynx: posterior wall injected; tonsils: removed	Polymorphonuclears, 40; lymphocytes, 10; eosinophils, 50	Ragweed 2 plus; house dust 4 plus; duck feathers 2 plus; goose feathers 2 plus
2 Mr. R. McC. 48 years	Alternating nasal obstruction to breathing; frequent head colds; had asthma until twelve years ago; sneezed considerably; no seasonal variation; had colitis	Nose: mucous membrane inflamed; septum deviated to left anteriorly; septal spur on both sides; turbinates normal; frontals clear; antrums slightly dark on transillumination; pharynx: injected; tonsils: removed	Polymorphonuclears, 0; lymphocytes, 60 eosinophils, 40	Refused
3 Mr. M. B. 46 years	Nose stuffed continually; alternated from side to side; watery postnasal discharge; sneezing marked in morning; no seasonal variation; no itching of eyes, nose or mouth; had colitis	Nose: mucous membrane injected; septum straight; turbinates normal; frontals and antrums clear on transillumination; pharynx: posterior wall injected; tonsils: removed	Polymorphonuclears, 0; lymphocytes, 100; eosinophils, 0; repeated tests failed to show the presence of eosinophils	Wheat 4 plus; milk 3 plus; navy beans 4 plus; string beans 4 plus; lima beans 4 plus
4 Miss O. P. 23 years	Postnasal dripping, nasal congestion and sore throats for past one and a half years; sneezed markedly in mornings; itching and swelling of eyes occasionally	Nose: mucous membrane injected; septum showed spur along right base; turbinates normal; sinuses clear on transillumination; pharynx: posterior wall injected; anterior pillars of tonsils showed no injection; no pus on pressure	Polymorphonuclears, 0; lymphocytes, 45; eosinophils, 55	Orris root 3 plus; house dust 4 plus; goose feathers 3 plus
5 Mr. W. H. 55 years	Nasal obstruction both nostrils for past ten years; polypi had been removed from both nostrils several times; sneezes considerably; itching of nose present; thin watery nasal discharge	Nose: mucous membrane pink; septum straight; polypi in each middle meatus; anterior tips of each middle turbinate undergoing polypoid degeneration; frontals and antrum clear on transillumination; pharynx: posterior wall normal; tonsils: cryptic	Polymorphonuclears, 0; lymphocytes, 40; eosinophils, 60	Orris root 3 plus; goose feathers 4 plus
6 Mr. B. P. 16 years	Frequent attacks of nasal congestion with watery discharge; sneezed considerably; pharyngitis also present	Nose: mucous membrane inflamed; septum straight; turbinates normal; watery discharge present; frontals and antrums clear on transillumination; pharynx: showed follicular injection; tonsils: out	Polymorphonuclears, 5; lymphocytes, 40; eosinophils, 55	Pecans 4 plus; goose feathers 2 plus; dust (house) 3 plus; cow dander 2 plus
7 Mr. L. P. 25 years	Nasal congestion and watery nasal discharge; itching of eyes; began in April and lasted about six weeks; same type of attack as last year	Nose: mucous membrane pale and watery; septum deviated to right, and in pressure contact with right middle turbinate; sinuses clear on transillumination; pharynx: posterior wall normal; tonsils: out	Polymorphonuclears, 5; lymphocytes, 39; eosinophils, 56	Not tested
8 Miss V. H. 22 years	Pain and smarting of eyes for past two weeks; first attack (April); had hay fever last fall; nose congested and watery	Nose: mucous membrane pale and watery; polypoid degeneration of both middle turbinates; watery secretion present in each nostril; frontals and antrums clear on transillumination; pharynx: posterior wall normal; tonsils: out	Polymorphonuclears, 5; lymphocytes, 45; eosinophils, 50	House dust 4 plus; bermuda 4 plus; ash 3 plus; ragweed 4 plus; orris root 3 plus; egg 4 plus

9 Mrs. C. E. 58 years	Profuse watery discharge from nose on awakening in the morning; frequent colds in head and sore throat; no itching of eyes or nose	Nose: mucous membrane pale; septum straight; turbinates normal; frontals and antrums clear on transillumination; pharynx: posterior wall injected; tonsils: out	Polymorphonuclears, 5, 0; lymphocytes, 95, 100; eosinophils, 0, 0 Polymorphonuclears, 20, 10; lymphocytes, 80, 20; eosinophils, 0, 70	Tests refused
10 Master B. B. 5 years	Frequent head colds; profuse watery nasal discharge for past year	Nose: mucous membrane pale and watery; turbinates enlarged; antrums clear on transillumination; pharynx: posterior wall normal; tonsils: had been removed	Polymorphonuclears, 0; lymphocytes, 0; eosinophils, 100	House dust 3 plus; orris root 2 plus; chicken 2 plus
11 Master V. F. 2 years	Stuffy nose with mucoid discharge continually for past year; also had eczema	Nose: profuse mucoid discharge present in each nostril	Polymorphonuclears, 0; lymphocytes, 20; eosinophils, 80	Oatmeal 3 plus; feathers 2 plus
12 Mr. A. W. 22 years	Nasal congestion with watery discharge all year; no itching of eyes, nose or mouth; had hay fever in late summer, lasting until frost	Nose: mucous membrane pale; both middle turbinates undergoing polypoid degeneration; septal spur along right base; frontals clear; both antrums dark on transillumination	Polymorphonuclears, 0; lymphocytes, 95; eosinophils, 5; blood showed 9% eosinophils	Tests refused
13 Mr. J. W. 20 years	Had hay fever from August until October; no trouble with nose except at that time	Nose: mucous membrane pale pink; septum deviated to left; turbinates normal; left antrum dark on transillumination; pharynx: posterior wall not injected; tonsils: out; small piece present in left fossa	Polymorphonuclears, 9; lymphocytes, 58; eosinophils, 33	Giant ragweed 3 plus; short ragweed 2 plus
14 Master W. M. 12 years	Nose congested from May till frost in fall; sneezing marked and eyes itched and watered in this period	Nose: mucous membrane pale and watery; septum straight; turbinates congested; discharge, thin mucoid; pharynx: moderately injected; tonsils: out	Polymorphonuclears; lymphocytes; eosinophils	Bermuda 4 plus; short ragweed 3 plus; giant ragweed 3 plus
15 Mr. C. R. 30 years	Alternating stoppage of nostrils for past three years, accompanied by sore throat; no seasonal variation; eyes become inflamed and nose congested immediately after swallowing a drink of whisky	Nose: mucous membrane injected; small septal spur along left base; turbinates normal; x-ray film of sinuses showed all sinuses clear; pharynx: posterior wall injected; tonsils: out	Polymorphonuclears, 11; lymphocytes, 89; eosinophils, 0; repeated smears had failed to show eosinophils	Tobacco 4 plus; orris root 2 plus; water hemp 3 plus; corn 3 plus
16 Miss B. P. 12 years	Itching of nose and watery discharge all year; worse from August till frost	Nose: mucous membrane injected; septum straight; turbinates normal; frontals and antrums clear on transillumination; pharynx: posterior wall normal; tonsils: out	Polymorphonuclears; lymphocytes; eosinophils	Orris root 3 plus; house dust 3 plus; ragweed 2 plus
17 Miss B. H. 25 years	Polypi had been removed from nose on twelve different occasions in past seven years; sneezed considerably; right nostril almost completely closed; worse in late summer	Nose: mucous membrane pale and watery; right nostril almost completely closed with polypi; polypoid degeneration of right inferior and middle turbinates; pharynx: posterior wall normal; tonsils: cryptic	Polymorphonuclears, 0; lymphocytes, 0; eosinophils, 100	House dust 3 plus; orris root 2 plus; giant ragweed 2 plus; short ragweed 3 plus
18 Master W. E. 5 years	Nasal congestion and discharge for past four years; frequent gastro-intestinal upsets diagnosed by pediatrician as allergic; grandmother had sensitization	Nose: mucous membrane injected; turbinates congested; pharynx: posterior wall injected; tonsils: removed	Polymorphonuclears, 0; lymphocytes, 75 eosinophils, 25	Tests refused
19 Miss B. W. 10 years	Nose stuffed and headaches for past three months; sneezing marked in mornings; profuse watery nasal discharge; swelling and itching of eyes at times; father had asthma	Nose: mucous membrane pale pink; turbinates not congested; septum showed spur along right and left base; frontals clear; antrums moderately dark on transillumination; pharynx: posterior wall normal; tonsils: out	Polymorphonuclears, 40; lymphocytes, 10; eosinophils, 50	Orris root 3 plus; duck feathers 2 plus

Owing to the relatively small number of cases and the fact that sufficient time has not yet elapsed to desensitize the patient to the agent found on testing, we prefer not to draw any definite conclusions as to the relief of symptoms, but merely to show the relationship existing between allergy as proved by the dermal tests and exhibited by the eosinophilia occurring in secretion from the nasal mucosa.

Case 15 is worthy of mention. When the nasal examination showed a red, congested mucous membrane, it was assumed that a postnasal dripping was responsible for the pharyngitis. The patient was treated with Dowling packs over a period of months without any visible improvement. His left antrum was irrigated, owing to the fact that it was dark on transillumination, without any benefit or the demonstration of any mucus or pus. Repeated nasal smears had failed to show an eosinophilia. He was then referred to the allergist, who found him sensitive to tobacco, orris root, water hemp and corn. His condition improved somewhat with the elimination of orris root and corn liquor. He did not stop smoking until he was tested again by another allergist in a neighboring city with the same findings; after he stopped smoking there was almost immediate relief from the nasal congestion and pharyngitis.

SUMMARY

The ages of the patients in the series varied from 2 to 55 years. The ratio between male and female patients was about equal.

The inhalants were the most common offenders, house dust appearing six times, orris root eight times, ragweed six times and feathers five times, followed by various other grasses and pollens with less frequency.

No one food stood out conspicuously in the results of testing.

Contacts causing allergy with symptoms referable to the nose and eyes did not appear in our series of cases.

The most frequent symptom complained of was obstruction to nasal breathing, due to nasal congestion, accompanied by a watery nasal discharge. Sneezing was the next in frequency.

Seasonal variation was found in six cases. In the remainder there were symptoms referable to the nose the year around. Exacerbations on exposure to dust were marked.

Seven patients had other conditions suggestive of an allergic cause, such as eczema, asthma, migraine or spastic colitis.

Contrary to the statement usually made that allergic patients show a pale and water-logged nasal mucosa, we found that the color varied from a pale pink to a deeply injected red. It was only in cases in which the symptoms had been present over a period of months that the so-called textbook picture of the pale and boggy mucosa was found.

Polypi were found in five cases. Some authorities state that these are the results of a rarefying osteitis of the underlying bone. However, since polypi appear in other organs of the body which have no bony boundaries, it would seem more logical to assume they are the result of a local inflammatory process and as such could be the result of allergy. This view has been expressed by others. In only a few cases was a dark frontal or maxillary sinus found on transillumination.

The percentage of eosinophils found on examination of the slides varied from 5 to 100. Several cases which because of the history convinced us of their allergic nature required from six to eight slides, taken on as many occasions, to demonstrate the presence of an eosinophilia. We have found the count on the same person to vary markedly within a relatively short time (twenty-four hours). This may be due to either one or both of two factors: first, an actual difference in the percentage of the various types of cells present did exist and, second, only 100 cells were counted. The cells tend to lie in groups of from 3 to 10 or more, and the field examined may not be a true index of the total number present.

CONCLUSIONS

1. Many cases of allergy in which the patients consult the rhinologist for relief are left undiagnosed, owing to a lack of careful history taking.

2. A cytologic examination of smears taken from the nasal mucosa is an invaluable aid in the diagnosis of allergy, as manifested by rhinologic symptoms.

3. Numerous slides may have to be taken to demonstrate an eosinophilia, since at times eosinophils appear to be temporarily absent in allergic cases.

CONSTITUTIONAL DEAFNESS

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ETIOLOGY [1]

In 1925, Drury [2] showed that in cases in which anatomic defects or concrete pathologic changes could be eliminated the level of aural acuity as measured by audiometric devices was an index of the nutritional level of the individual. Little attention was paid to this statement. It was again brought to my attention in "Vital Function Studies," by Rowe and Drury.[3] This is the most important generalization relative to deafness of constitutional origin that has yet been uttered. Taking due cognizance of the importance of this dictum, the related data in the literature and the accumulated material at hand, the etiology of this condition may be set forth as follows:

Predisposing Causes	Exciting Causes
Heredity	Hepatic
Environment	Hepatic and Pancreatic
	Endocrine

PREDISPOSING CAUSES

Heredity.—Practically all of the literature on the subject of heredity is based on the assumption that the cases with which the authors dealt were cases of otosclerosis. In this treatise, otosclerosis is not mentioned, except when reference is made to the work of others who have thus classified their cases. I reiterate the statement which I made in an article entitled, "Deafness, Hepatic Dysfunction, Pancreatic Insufficiency: A Clinical Entity." [4]

As will be noticed, no effort is made to classify the cases according to the standard nomenclature. The author does not distinguish the cases by saying these are cases of nerve deafness, chronic catarrhal deafness, or otosclerosis so called. The view is assumed here that the character of the hearing curve and other signs found in deafness should not be used as a means of making a pathological diagnosis. The difference in hearing curves in varous cases of deafness are due to the accidental location of the lesion or the vagaries of the anatomy of the hearing

1. In this article free use has been made of material and text from Duel, A. B.: Otosclerosis, New York, Paul B. Hoeber, Inc., 1929.

2. Drury, D. W.: Tests of Hearing of Five Hundred Average Ears by the Audiometer 2-A, Arch. Otolaryng. **1**:524 (May) 1925.

3. Rowe, A. W., and Drury, D. W.: Vital Function Studies, J. A. M. A. **98**:1539 (April 30) 1932.

4. Gottlieb, M. J.: Ann. Otol., Rhin. & Laryng. **41**:523 (June) 1932.

organ. The best one can say is that the patient is deaf and if the cause can be found it should be named accordingly.

In the literature from time to time reports of cases of deafness have been made which include histologic studies of the temporal bone. In some cases definite lesions of the bone were found, and in such instances a diagnosis of ostosclerosis is tenable and proper. Bony lesions, as described by a number of investigators, were found in many instances at their usual site of occurrence, namely, in the region between the oval and the round windows, but in other cases of deafness such lesions were discovered in other parts of the bony labyrinth and could not possibly have been the cause of the deafness. Many of the cases studied occurred as a family disposition, and among such cases several congenital deaf-mutes were encountered. Cases of labyrinthine deafness were also found in these families.

For the sake of emphasis I refer to Hammerschlag,[5] who maintained that the degenerative-atrophic processes in the nerve and the nerve end terminals that are found in congenital deafness are probably identical with those found in "otosclerosis." The bony process in the labyrinthine capsule, characteristic of otosclerosis, is found also in some cases of congenital deafness. There are families in which hereditary deafness and otosclerosis are found in association. All these reasons justify the question whether hereditary deafness and otosclerosis may not properly be interpreted as different manifestations of the same pathologic process. Hammerschlag presented two genealogies, in one of which congenital deaf-mutism and partial congenital deafness were present in a family through five generations; all members of this family who were either totally or partially deaf were affected from birth. It is evident that congenital deaf-mutism and partial deafness that is also congenital represent only different degrees of the same condition. This family also showed various psychoses and other diseases of the central nervous system.

The second genealogy showed a family affected by various types of deafness. Two sisters, stated to have had normal hearing, had seven and six children, respectively. Of the seven children of the older sister, the oldest, a son, gradually became deaf at an advanced age; the second child, a girl, was a deaf-mute as the result of a fall in childhood it was claimed. Three normal children followed. The sixth child, when examined by the author, a woman of 59, presented findings typical of otosclerosis, complicated by advanced involvement of the acoustic nerve. (Rinne's test was negative, but bone conduction was no longer prolonged.) The younger sister had six children, one of whom, a son, gradually became completely deaf at an advanced age.

5. Hammerschlag, V.: Ztschr. f. Ohrenh. **59**:315, 1909.

One type of deafness encountered in this family therefore manifests itself as a condition appearing in middle life and leading progressively, in the course of years, to an almost complete loss of hearing. According to one case examined, this auditory disturbance may, with a fair degree of certainty, be designated as otosclerosis. The second type of deafness in this family, the deaf-mutism of a female member, requires a brief discussion. According to the history, the deafness of the patient would be designated as acquired, for it was positively stated that hearing was lost after a fall. This woman later married a man of normal hearing and had two children, deaf-mutes from birth. It may therefore be regarded as highly probable that in this case, as occurs often, the traumatism merely aggravated a congenital condition.

In 1910, Hammerschlag [6] again referred to the genealogy discussed in his contribution of 1909, which showed the association of hereditary degenerative deaf-mutism and otosclerosis in the same family. He maintained that otosclerosis and hereditary degenerative deaf-mutism are different manifestations of the same hereditary process, on the following grounds, as suggested in his previous article: 1. The degenerative processes in the nerve and the nerve terminals found in congenital deafness (deaf-mutism) are probably the same as those found in otosclerosis. 2. The characteristic changes in the bone in otosclerosis of the labyrinthine capsule are found in some cases of congenital deafness. 3. There are families in which hereditary deafness and otosclerosis are associated.

He pointed out elsewhere that progressive labyrinthine deafness is also based on a congenital disposition and is regarded by many otologists as definitely hereditary. In a case of progressive labyrinthine deafness reported by Manasse, an isolated new-formed focus of spongy bone was found in the internal auditory meatus in addition to the typical pathologic changes in the nerve. The findings indicate that progressive labyrinthine deafness is a primary degenerative atrophy of the acoustic nerve, which may not be manifest until later in life but which in persons with a constitutionally inferior auditory organ is manifest in early life. The author, therefore, does not hesitate to state that hereditary degenerative deafness (deaf-mutism), progressive labyrinthine hardness of hearing and otosclerosis merely represent different manifestations of a genetically uniform disease.

Mayer (1911) [7] developed his theory that the focal disease in the petrous bone characteristic of otosclerosis is produced by local circulatory disturbances in the terminal ramifications of the arteries of the bone. He noted that this also indicates a further resemblance between oto-

6. Hammerschlag, V.: Monatschr. f. Ohrenh. **44**:769, 1910.

7. Mayer, O.: Monatschr. f. Ohrenh. **45**:421, 1911.

sclerosis and osteitis fibrosa, as Recklinghausen ascribed the latter to circulatory disturbances resulting from an irritation of the vasomotor nerves and leading to repeated congestion of the bone marrow. Clinical studies show that some patients with otosclerosis present symptoms indicating vasomotor disturbances.

Aside from these functional disturbances of the vasomotor apparatus, organic changes in the blood vessels may also be the cause of the circulatory disturbances that are the basis of the otosclerotic changes. The part played by arteriosclerosis as the cause of the vascular constriction in otosclerosis is suggested by anatomic findings. Autopsy reports show that in a number of cases of otosclerosis arteriosclerotic (atherosclerotic) changes of the blood vessels are found. Furthermore, the same general diseases that are considered to be etiologic factors in arteriosclerosis are also regarded as the cause of otosclerosis. Special mention is made in this connection of the acute infectious diseases, more particularly scarlet fever, typhoid fever, diphtheria and influenza. It is especially noteworthy that the majority of juvenile and presenile cases of arteriosclerosis are referred to preceding infectious diseases. In view of the known tendency of certain infectious diseases to produce sclerosis only in definite vascular areas, it is possible that there are infectious diseases which give rise to isolated sclerosis in the bone of the pyramid.

The question of whether or not otosclerosis can be produced or aggravated by traumatism has recently been raised, and in view of the known origin of atherosclerosis through traumatism, Mayer regards traumatism as entirely possible as an indirect cause of otosclerosis. Vasomotor neuroses may also follow on traumatism. Syphilis may enter into consideration as an etiologic factor, through the causation of vascular changes, but it is evidently not a frequent cause of otosclerosis.

The connection of the labyrinthine atrophy, noted in otosclerosis, with the bone disease is not clear. On the basis of histologic investigations, Mayer has arrived at the view that pathologic conditions of the bone and labyrinthine atrophy are not directly independent, but that both conditions are referable to the same fundamental cause, namely, damage to the vascular system. The histologic changes of the stria vascularis resemble those usually seen in arteriosclerosis, and the atrophy of Corti's organ is probably caused by the atrophy of the striae. Since the labyrinthine atrophy, as well as the focal disease of the bone in otosclerosis, can be produced by vascular disturbances, it is readily understood that both conditions, that of the bone and that of the labyrinth, may occur simultaneously in otosclerosis. In cases in which no organic vascular changes are present, but vasomotor disturbances enter into consideration as the cause of the otosclerosis, the labyrinthine atrophy may perhaps be caused through vasomotor trophic disturbances.

The heredity of otosclerosis is apparently due to transmission of the cause of the disease, for heredity is known to play an important part in arteriosclerosis as well as in the vasomotor neuroses.

Ferreri [8] stated that in recent years the theory that otosclerosis is due to some form of autointoxication has gained ground. It has been stated that in certain countries, such as England and central Italy, where gout and gouty diathesis are of frequent occurrence, primary otosclerosis also is frequent. But the majority of cases of otosclerosis observed with gout are not due directly to the gout, but to the state of autointoxication that exists in these patients because they do not eliminate certain metabolic products from the blood. It may be admitted that such autointoxications are latent and hereditary, so that otosclerosis does not develop until a period of special strain on the organism, as during the period of the development of the skeleton in rachitic subjects or during the period of pregnancy and lactation in women with osteomalacia. In some cases of congenital deaf-mutism, the loss of hearing results from pathologic changes in the labyrinthine capsule that originated during fetal life owing to some maternal autointoxication (syphilitic or otherwise). Otosclerosis is sometimes undoubtedly associated with syphilis. Rickets and osteomalacia Ferreri found frequently associated with otosclerosis.

Bauer and Stein [9] reported a study of the general condition and family history of twenty-six patients with otosclerosis. They found that these patients showed many evidences of constitutional abnormalities and degenerative stigmas. The family histories showed that there were present in the family in some cases not only otosclerosis but also diseases known to develop on the basis of constitutional inferiority (diabetes, premature arteriosclerosis, chlorosis and similar conditions). According to present knowledge, only the tendency to otosclerosis can be regarded as inherited. While evidence of deafness in some members of the family was found in several of the cases reported, the family history of most interest was that of a girl, aged 19, whose father, uncle and grandmother on the paternal side had labyrinthine deafness as proved by otologic examination; on the maternal side, a sister of the grandmother had otosclerosis, and three half-sisters (same father) were congenitally deaf. This family history would support the theory of Hammerschlag that hereditary degenerative deafness, progressive labyrinthine deafness and otosclerosis are only different forms of a single genetic disease.

It can readily be seen that the statements made by the foregoing investigators lead to the conclusion that the bony changes in the

8. Ferrari, G.: Arch. internat. de laryng. **30**:1, 1910; translation, Ann. Otol., Rhin. & Laryng. **20**:94, 1911.

9. Bauer, J., and Stein, C.: Ztschr. f. ang. Anat. **1**:546, 1914.

labyrinthine capsule and the degenerative changes in the highly specialized tissue of the cochlea are expressions of a common etiology; also, that congenital degenerative deafness (deaf-mutism) falls into the same category as, and is probably initiated by, some toxemia that occurred in the mother during the period of gestation.

The theory, propounded by Mayer,[7] that disturbances of the circulation of the bony labyrinthine capsule and the organ of hearing itself may be of etiologic significance assumes importance in view of the fact that the different types of terminal circulation of the cochlea do occur in man, and there is no reason to believe that such differences may not also exist in the bony labyrinthine capsule. It is my belief that heredity plays its most important part at this point. Conditions of unsatisfactory terminal circulation predispose the patient to deafness when other organs or systems become deranged. I made the following statement in an article entitled, "Deafness Due to Pancreatic Insufficiency." [10]

> The incidence of deafness associated with pancreatic dysfunction is so great that it cannot be disregarded. Its association is not accidental. However, all cases of pancreatic disease do not go deaf. The explanation for this may be found in studying the difference in anatomy of various individuals. These differences are transmitted from parent to offspring. Polydactylitis occurs in families. Impacted wisdom teeth are more likely to occur in issues from parents whose molars were also impacted. The circulation of the cochlea varies in individuals. Nabeya has shown that there are three types of circulation in the cochlea of man. In the first kind, the arteria propria cochleae is practically a terminal vessel from its source, there being only a few minute arborizations between it and the arteria vestibulocochleae in the basal coil. A person with such a cochlear circulation is more liable to become deafened than one who has the second kind or the third kind of blood supply. There is no doubt that during toxemia of any origin the sensitiveness of nerves is diminished, and when an organ is especially vulnerable greater damage will result. It is possible that we inherit the type of circulation that our ancestors possessed, and this may explain the frequency of hereditary deafness. That hearing losses are frequently encountered in cases suffering with circulatory diseases is suggestive of a relation between auditory acuity and blood-vessel integrity. This idea is further encouraged when, on investigating the detailed symptoms of the cases in consideration, we find that the tinnitus is made louder and more annoying, and that the hearing is less acute following coitus. Furthermore, following a short interval after awakening from sleep, the tinnitus is resumed with greater force than at any other time during the day, and this lasts for a short time. There is no doubt that profound changes occur in the circulation just prior to and following sexual intercourse and during the transition from sleeping to waking.

This idea is encouraged further when one considers that Wever and Bray [11] have demonstrated that in the experimental animal the organ of hearing ceases to function as an instrument for conveying sound impulses as soon as the supply of blood to the cochlea is shut off.

10. Gottlieb, M. J.: Tr. Am. Acad. Ophth. **35**:386, 1930.

11. Wever, E. G., and Bray, C. W.: J. Exper. Psychol. **13**:373, 1930.

Furthermore, Wegel [12] has shown by an ingenious method that the tinnitus in his own ear is of circulatory origin. This view is also supported by E. M. Josephson [13] in a philosophic consideration of the origin of tinnitus. I subscribe to this idea and believe that tinnitus in cases of deafness of constitutional origin is due to constriction of the blood vessels supplying the cochlea. This constriction may be a spasm, an actual adventitious thickening or a narrowing of the vessel due to circumferential pressure from exudates or inflammatory products in the vicinity of such vascular channels. Spasms of retinal vessels (Agatston [14]) accompanied by temporary impairment of sight is common, and there is no reason why such a condition cannot pertain to the ear as well. By the same token, impairment of hearing can occur due to either spasm or permanent stenosis of the cochlear artery from any cause.

Environment.—Drury [15] noted that in 40 per cent of his patients with otosclerosis there was a definite family history of deafness. Heredity plays a very important part in the etiology of otosclerosis, although data are lacking to warrant any generalization of the existence of a mendelian formula. A like tendency toward transmissibility is demonstrable in disturbances of the endocrine glands.

There is no doubt that in a high percentage of cases of deafness there is a familial or hereditary history of deafness. On the other hand, single cases of difficulty in hearing do occur in families that on detailed search reveal no other cases.

Diabetes is prevalent in some families, while in others it does not occur; isolated cases, however, are encountered. This applies to endocrine diseases and probably to other ailments, such as cardiac and renal disturbances. Deafness occurs in cases of endocrine disease, especially in cases of hypofunction of the pituitary and thyroid glands, but all cases in which lowered function of the pituitary and thyroid glands is a predominant feature are not associated with difficulty in hearing. This is explained by the assumption that there exists a vulnerable circulation of the cochleae in some persons, while it is not so easily affected in others. The hearing curve of practically all persons suffering with disease of the gallbladder, diabetes and circulatory diseases shows a drop, especially in the high notes. In cases of recent onset the depreciation of hearing is less than in those of long standing. Due consideration is given to the fact that in the aged there is also a drop in acuity of hearing in the high tones.

12. Wegel, R. L.: A Study of Tinnitus, Arch. Otolaryng. **14**:159 (Aug.) 1931.
13. Josephson, E. M.: Spasm of the Auditory Accommodation Mechanism, Arch. Otolaryng. **13**:166 (Feb.) 1931.
14. Agatston, S. A.: Am. J. Ophth. **13**:309 (April) 1930.
15. Drury, D. W.: Acta oto-laryng. **10**:90, 1926.

The fact that deafness occurs in families does not mean that the condition is hereditary or familial. Little consideration has been given to the influence of environment. It is admitted that the vulnerability of the cochlea is greater in some than in others. Such vulnerability is a transmissible trait; the exciting cause, however, may be engendered by the mode of life common to the family group. I have found cases of dysfunction of the liver and pancreatic insufficiency existing in one family. These were also cases of deafness. This association also applies to endocrine disturbances. The following passage [16] serves to express a summary of the idea:

> The human organism enters this life, as St. Augustine puts it, *inter faeces et urinam,* and is immediately infected with the bacteria of the intestinal and genito-urinary system of the mother. In six or eight hours, the child is suckled and exposed to the skin flora of the mother. In due time, the mother or father and other relatives contribute their portion of bacteria to the mouth and respiratory tract by means of osculation. The method of preparing food is usually handed from mother to daughter through generations. If the family's economic condition remains the same for a considerable length of time, the question of general environment, such as sunshine, ventilation, cleanliness, and sufficiency of rest and mental state (contentment or disquiet), assumes importance. If there is an improvement in the economic condition of the family, however, the only change that occurs is in the general and mental environment, rather than the personal. With such potent factors as those enumerated, why is it not possible for similar bodily ailments to develop in one family, as compared to other families having no such maladies?

EXCITING CAUSES

General Considerations.—The symptoms common to all cases of deafness of constitutional origin are progressive bilateral impairment of hearing, tinnitus and vertigo. In women, the tendency of deafness to start during pregnancy or lactation in some cases or to progress at an accelerated pace during this period is well known. Inflation in such cases at times produces improvement in hearing; at other times there is improvement in some frequencies and depression in others. There is no consistency in the response to inflation by the organ of hearing. I have given this matter careful consideration in a previous communication.[4]

> The effect of inflation on the hearing curve in these cases is peculiar, insofar as at times the hearing was improved in most of the frequencies after inflation. On the other hand, taking the same patient at another time, the hearing was decidedly depressed in all frequencies or only in a few of the tones, or the hearing for some tones was improved while other tones were depressed. In other words, inflation, in the type of case under consideration, has no diagnostic value. It has value, however, in cases of impaired hearing due to scarring from a previous middle-ear suppuration. When, in such a case, constitutional factors further depress the hearing, it has diagnostic and therapeutic value. The question of inflation as

16. Gottlieb, M. J.: Laryngoscope **39**:485 (Aug.) 1929.

a general proposition deserves some consideration in view of the recent work of Hughson and Crowe,[17] who, using the method described by Wever and Bray,[11] have determined that the membrane of the round window acts as a safety valve in absorbing about 50 per cent of the sound coming to the ear, as a protection for the cochlea. When the membrane of the round window is compressed, it increases the auditory acuity for sounds from 512 to 4,096. It may be adduced from this that most of the improvement in hearing which is experienced as the result of inflation is due to a sustained increase of air pressure after inflation. As soon as the air leaks out of the Eustachian orifice, or is absorbed, the hearing returns to its original acuity. This is the reason why ears that have wide-open Eustachian tubes show no improvement on inflation. The majority of the cases herein reported were easily inflated.

Many students of nonsuppurative deafness in which no local reason can be found for the condition have finally been compelled to conclude that the exciting cause is constitutional. Much has been written on the subject. For the purpose of emphasis the following authorities are cited:

Gradenigo[18] regarded anomalies of embryonic development as the essential predisposing cause of sclerotic otitis; other pathogenic factors can be recognized in the majority of cases. In women he found that the disease often manifests itself, or is seriously aggravated, during pregnancy and the puerperium, so that each childbirth is followed by definite diminution of hearing. In both sexes, conditions are usually found that indicate abnormal vascular irritability: rise of blood to the head, vertigo of Ménière's type, circulatory disturbances, edema and similar conditions. A number of Gradenigo's patients with sclerotic otitis were examined by Giovanni in regard to the vascular system; a more or less exaggerated vascular irritability was found in these cases. Gradenigo has noted also that the subjective noises and deafness of sclerotic otitis are increased by all causes favoring congestion in the head.

Heiman[19] distinguished between catarrhal and sclerotic otitis, but stated that the same causes may produce either form. He found, however, that the causes of catarrhal otitis are more often local; those of sclerotic otitis, more often general. Heredity is an important factor in sclerotic otitis. Pregnancy is also an important etiologic factor, especially if profuse hemorrhage occurs in labor. Among other conditions that may be etiologic factors in sclerotic otitis the author names anemia and chlorosis, chronic rheumatism and gout, leukemia, diabetes, syphilis, oxerexertion and abuse of alcohol.

17. Hughson, W., and Crowe, S. J.: Function of the Round Window, J. A. M. A. **96**:202 (June 13) 1931.

18. Gradenigo, G.: Riv. veneta di sc. med. **7**:417, 1887.

19. Heiman, T.: Wien. klin. Rundschau **12**:765, 782, 797, 815 and 831; Monatschr. f. Ohrenh. **43**:761, 804 and 909, 1898.

Dickie [20] concluded from his clinical study of otosclerosis that it is one manifestation of a fault in metabolism, a uric acid toxemia due to the retention of this metabolic product in the system, a toxemia that attacks the individual "in his most vulnerable point, the labyrinth."

Katz [21] stated that he was convinced that characteristic bony changes in otosclerosis are based on a constitutional or dyscrasic, usually hereditary, disease, on the basis of which the temporal bone or the auditory organ enclosed in it represents a point of diminished resistance acted on by a series of injurious factors.

Heiman, on the basis of his observations in the study of two hundred and twenty-three cases of otosclerosis and of a review of the literature, concluded that otosclerosis is a localization of a general disease or a dyscrasia in the auditory organ. The basis of the general condition is an acute or chronic nutritional disturbance, leading to pathologic changes in the blood. The correctness of this interpretation is confirmed by the uselessness of all local therapy and the general benefit that results when the patient's general condition is treated. It is undeniable that otosclerosis is hereditary, but heredity is not the only cause.

Bryant [22] stated that the general etiologic factor in otosclerosis may be toxemia of any kind: autointoxication; rheumatic, gouty or syphilitic conditions; intoxication by infectious diseases, and puerperal and menstrual disturbances; also that the sympathetic nerves supplying the upper respiratory tract become the cause of chronic interstitial otitis (otosclerosis) by their functional instability and by their toxic derangements in chronic alcoholism, chronic tobacco poisoning and similar pathologic processes. Unbalanced internal secretions that affect the ear are disturbances in the secretions of the pharyngeal tonsil, the faucial hypophysis, the suprarenal and the thyroid glands and the faucial tonsil.

According to Gray's [23] theory, "the primary fault" in otosclerosis is an inherent defect in the living cells of the organ of hearing. Anemia, pregnancy, syphilis, toxic products in the blood, exposure to extreme cold, abnormal internal secretions, other general constitutional conditions or pathologic changes in the middle ear are not necessary for the occurrence of otosclerosis. Such conditions may, however, play an active part in making manifest a tendency that would otherwise have remained latent. The general or local conditions in the middle ear and eustachian tube that may evoke the variation or the manifest symptoms may be different in different cases. In many cases the innate tendency of the cells of the organ of hearing is sufficiently pronounced so that

20. Dickie, P.: Homaeop. Eye, Ear & Throat J. **9**:330, 1903.
21. Katz, L.: Arch. f. Ohrenh. **68**:122, 1906.
22. Bryant, W. S.: Ann. Otol., Rhin. & Laryng. **19**:232, 1910.
23. Gray, A. A.: Tr. Am. Acad. Ophth. **16**:9, 1911.

otosclerosis will become manifest without any other discoverable pathologic condition, local or general. The mere pathologic processes of growth and repair are sufficient to make the tendency manifest.

Amadon (1912) [24] described his pathologic findings in a case of otosclerosis. In this case he noted not only that there was a family history of deafness but also that the patient was "rheumatic." He stated that otosclerosis is often associated with such conditions as anemia, arteriosclerosis or some of the arthritides, the etiology of which is not clearly understood. The pathologic changes are closely analogous to those in arthritis deformans. Amadon is of the opinion that otosclerosis is a disease due to locally disturbed nutrition. The labyrinthine capsule is a bone of unique structure. No bony structure in the body undergoes so little change during the course of a lifetime as does this capsule, and it always shows vestiges of the embryonic capsule in which it is laid down. The bone is immature, and tissue of immature type is "very sensitive to nutritive disturbances." Amadon believes that the bony metaplasia in otosclerosis is not due to any specific or local exciting cause, but to general nutritional disturbances acting on a bony tissue with unique histologic characteristics.

Raoult,[25] who maintained that otosclerosis is due primarily to neuritis, stated that this neuritis, as other forms of peripheral neuritis may be caused by different factors: infections, intoxications, autointoxications, pregnancy and anemia.

Bauer and Stein [9] presented a study of the constitutional characteristics of twenty-six cases of otosclerosis in eighteen women and eight men ranging in age from 16 to 52 years. The importance of constitutional factors in the etiology of otosclerosis is indicated by the well known predilection of the disease for the female sex and its extremely frequent onset at the time of puberty. The influence of the constitutional factor is furthermore suggested by the fact, emphasized by numerous writers, that in otosclerotic patients systems or diseases are not infrequently encountered that are based on an abnormal general condition such as, for example, chlorosis, gout, uric acid diathesis, rachitis, osteomalacia, vasomotor disturbances, trophic neurotic processes and hypertrophy of the lymphatic pharyngeal ring. Finally, the definite statements of some patients, referring the beginning of the disease to a traumatism or a psychic injury, also point to an abnormal mode of reaction in the individual patient based on a pathologic constitution of the organism.

Systematic examination of the twenty-six patients in this series showed the following symptoms of an abnormal constitution, arranged

24. Amadon, A. M.: Laryngoscope **22**:927, 1912.

25. Raoult, A.: Bull. d'oto-rhino-laryng. **15**:233, 1912.

in the order of frequency: (1) anomalies of the vasomotor nerves (dermographism of variable intensity and quality and acrocyanosis); lability of heart action, as well as respiratory irregularities of the pulse; Aschner's bulb-pressure reflex (retardation of the pulse by pressure on the closed eyeballs); Erben's pulse phenomenon (retardation of pulse on flexing the knees or low stooping); exaggerated tendon and periosteal reflexes, especially in the upper extremities; absence of the corneal and pharyngeal reflexes; lateral differences in the reflexes of the abdomen in such a way that the reflexes of the left side are weaker than those of the right or altogether absent; (2) anomalies of the sexual function; premature or delayed onset of menstruation, irregularity of menstruation, dysmenorrhea and hypoplastic genitals; (3) anomalies in the hairy covering of the trunk (female type of pubic hair in men and the male type in women), as well as hairs on the chest and thighs in female patients, and absence of axillary hairs; (4) anomalies in the size of the breasts or nipples; hyperplasia of the lymphatic pharyngeal ring; high palate; enteroptosis, struma; chlorosis; signs of past rickets; migraine; asthenic thorax, with eventually a floating tenth rib; marked dental caries, with early loss of teeth and abnormal position of teeth; shortened percussion sound over the sternal manubrium and slightly to the left of it, owing to bronchial glands or a hyperplastic thymus; rigid vessels in youthful persons; accidental systolic bruit over the pulmonary valve and accentuated second pulmonary sound in the sense of a constitutional inferiority of the circulatory apparatus, without any demonstrable sign of organic disease; striking varicosities of the lower extremities in young women who have never been pregnant; abnormal pigmentation suggestive of chloasma in the face, hyperdistensibility of the joints, especially the metacarpalphalangeal joints; remarkably slender, tapering fingers; habitual constipation; acrophagia; membranous colitis; Chvostek's facial phenomenon; lingua plicata; stigmatism, irregular astigmatism, flatfoot; Heberden's nodes, and united eyebrows.

With the series of degenerative stigmas the authors include Froeschel's symptom of decreased tickling sensation in the external auditory meatus, the missing secretion of cerumen, as pointed out by Bing, and the diminished perspiration observed by Hammerschlag. The authors repeatedly had occasion to observe the two first-named symptoms in the patients and to demonstrate the third symptom in one instance.

The foregoing signs, which are by no means to be considered as the symptoms of a disease, but which may be regarded as the expression of a faulty disposition of the organism, are sometimes found separately in persons who must be designated as entirely normal; probably hardly a person is free from one or the other of these symptoms. The noteworthy feature in the cases examined by the authors is the large number

of stigmas found in the same person. The frequency of congenital signs of degeneration and their constant occurrence in patients suffering from otosclerosis indicate in all circumstances the hereditary-degenerative disposition of the patients and explain the abnormal reaction of the organism of such persons toward irritants of all kinds. The culmination of degenerative signs, as observed by the authors in persons with otosclerosis, is undoubtedly such as to exclude an accidental coincidence. It is a known fact that a large number of different pathologic conditions develop on the soil of an abnormal constitution characterized by the summation of degenerative stigmas. Status thymolymphaticus is a predisposing factor for a great variety of diseases, for example, lymphatic leukemia, polyneuritis and Addison's disease. In the cases of otosclerosis examined, the frequency of degenerative stigmas is so evident as to justify the conclusion that the development of otosclerosis is favored by the presence of a degenerative constitution. All the factors that are credited with being of etiologic significance in otosclerosis, such as syphilis, pregnancy and infection of the nasopharynx, seem, according to my investigations, to elicit otosclerosis only in the presence of a degenerative constitution, as already described.

The symptom of a degenerative disposition demonstrable in all cases of otosclerosis is the lability of the circulation, which has its cause in the hyperirritability of the vasomotor nerves and in the lability of the heart action. The possibility pointed out by O. Mayer [7] must therefore be kept in mind; namely, that the lability of the vasomotor nerves should be credited with special significance for the origin of otosclerosis, all factors that specially affect these nerves entering into consideration as determining factors. Included are certain factors that have been mentioned by writers as etiologically important for otosclerosis, such as psychic emotions, traumatisms, bacterial toxins and infections damaging the vasomotor apparatus, as well as alterations in the endocrine balance such as manifest themselves at the time of puberty and in pregnancy. Only further investigations can guarantee the correct estimation and appreciation of all these etiologic factors in the production of otosclerosis.

Peltesohn [26] noted that as the importance of the endocrine glands is beginning to be realized, the hyperfunction or hypofunction of some of these glands is being considered as an etiologic factor in otosclerosis. Especially is the pituitary gland to be considered, because of its well known influence on the growth of bone and also because the symptoms of otosclerosis often manifest themselves during puberty and are aggravated in women by pregnancy and the climacteric period, during which the function of the pituitary gland is known to be disturbed. Peltesohn is of the opinion that the predisposing factor in otosclerosis is

26. Peltesohn, F.: Berl. klin. Wchnschr. **55**:252, 1918.

congenital foci of the bone in the labyrinthine capsule, which begin to proliferate only under a variety of conditions among which the author mentions puberty, pregnancy, puerperium, catamenia and senility (action of endocrine glands) and physical or psychic trauma, as was shown during the war. Furthermore, all infectious diseases cause this proliferation, such as acute and chronic otitis, rhinitis, intoxications (due to alcohol, nicotine, etc.) and the so-called dyscrasias, to which can probably be added also all diseases of the blood, such as anemia, chlorosis and leukemia.

The constitutional character of the disease is indicated by the bilaterality of the process, which, however, does not necessarily affect the labyrinth at the same points or to the same degree. As such causes must act before the congenital foci begin to extend, it is easily understood why otosclerosis manifests itself clinically at different ages. The first signs of otosclerosis may be evident as early as the tenth or the fourteenth year of life, while there are other cases in which symptoms do not occur until ten or twenty years later; some cases remain stationary for many years and the condition appears in only one ear; still others lead to hardness of hearing of extreme degree within a very short time. The beginning of otosclerosis depends on the time when the injurious exciting cause stimulates the energy toward growth in the foci congenitally present. The variability in the course of the disease is governed by the stimulus of the exciting cause and the extent and peculiar situation of the foci of the bone in the labyrinth.

Finally, Rowe and Drury, mentioned earlier in this treatise,[3] examined the hearing of 2,078 children between the ages of 9 and 17 and found that 276 had faulty hearing. Eighty-six of these had mental or physical defects of undetermined etiology. Forty-four finally presented themselves for more or less thorough examination, and the diagnostic analysis was as follows:

Endocrine disturbances:	
Pituitary	7
Thyroid	5
Unclassified	2
Not endocrine:	
Lesions of the central nervous system	17
Psychosis	1
Renal disturbances	1
Infectious diseases	6
Syphilis	1
Unclassified	4
Mental status:	
Retarded, established	6
Retarded, suggestive	16
Normal	6
Not examined	16

They state that the examination is by far incomplete. It is unfortunate that the other forty-two cases were not available for a diagnostic analysis. It shows, however, as is also suggested by the other references, that cases of bilateral deafness have other signs and symptoms which when elicited and properly evaluated lay the basis for a determination of the cause of the hearing defect. There is no doubt that "the level of aural acuity is an index of the nutritional level of the individual." In every case of nonsuppurative bilateral deafness in which there is no local pathologic defect other general objective and subjective abnormalities occur which when correlated lead the way to finding their cause as well as that of the deafness. From this standpoint, I do not hesitate to say that constitutional deafness is a preventable and curable condition within certain well defined limits. It can be prevented by the institution of yearly tests with the audiometer of all persons above puberty. A drop in hearing below the original level should be immediately followed by a complete subjective and objective survey and all abnormalities should be corrected if possible. Hardness of hearing is curable in the sense that in the early cases prompt response to treatment is the rule. In cases that have existed over five years complete recovery is not to be expected, although some improvement does occur or the progress of decline in hearing is definitely checked.

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ASSOCIATION OF ACUTE SINUSITIS AND ACUTE OTITIS MEDIA IN INFANTS AND IN CHILDREN

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It is only within the last few years that sinusitis in infants and in children has been receiving the interest and attention it deserves, and even now the importance of this most common of all infantile disease has not received due emphasis, for one still finds that many practitioners and even pediatricians are treating patients with conditions such as bronchitis, asthma, gastro-intestinal disturbances and otitis media without sufficient regard to the control of the frequently coexisting sinus infection. While it is perhaps generally accepted that sinusitis may be an important factor in pathologic disturbances in older children, there seems to be a reluctance to attach any great significance to sinus infection in infants, and many physicians still believe that the sinuses of infants are insufficiently developed to be of pathologic importance, despite the anatomic proofs to the contrary.

While the association of sinusitis with respiratory disturbances and general conditions, such as arthritis and nephritis, is generally recognized, the close connection between infections of the sinuses and those of the ear is not so well understood and accepted. This close association was recognized by Fowler,[1] who reported, in 1929, 100 cases of diseases of the ear in children, with an involvement of the sinuses in 86 per cent. The purpose of this communication is to point out the connection between acute otitis media and acute sinusitis and to emphasize the great frequency of sinusitis in infancy and its importance as an etiologic factor in the production of the otitis.

It has been well said that the child has not yet been born who has reached the age of 1 year without having an acute cold. After the observation of many hundreds of infants in the past ten years, I wish to enlarge this statement to the effect that no child has reached the age of 1 year without a sinus infection. In recent articles on sinusitis it has been stated rather reservedly that infection may be as frequent in children as in adults. In my opinion, sinusitis is much more common

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1. Fowler, E. P.: The Incidence of Nasal Sinusitis with Diseases of the Ear, Arch. Otolaryng. **9**:159 (Feb.) 1929.

in infants and children than in adults, for acute colds are more common in the young, and practically all colds of infants are in reality sinus infections. It has been further stated by some writers that sinus infections are increasing in frequency. This apparent increase is largely the result of colds being called by their right name (sinusitis). However, there may be an actual increase due to such factors as greater congestion of population, living indoors, a tendency to dietary indiscretions, reduction in weight, etc. Sinusitis in infants is as common as colds, because an infection of the nasal mucous membrane is almost certain to reach the mucous membrane of the sinuses. This occurs, firstly, because the ostium of an infant's sinus is large in proportion to the size of the sinus and, secondly, because sneezing, coughing, crying and vomiting cause a regurgitation of the unremoved secretions into the sinuses through the large ostia.

The chief factor not only in the production of sinusitis in infants but also in the prolongation of the disease is the lack of removal of the nasal secretions. I have many times observed with the nasopharyngoscope how the simple act of crying forces the secretions of the nasopharynx and the posterior part of the nose high up into the middle meatus and spheno-ethmoidal recesses. The same simple act, together with the anatomic peculiarities of the infant's eustachian tube, is responsible for the otitis media so commonly associated with sinusitis. The eustachian tube of infants being shorter, wider and more patulous than that of older children and adults, infection of the middle ear by this route is an easy and common occurrence. The infected drainage from the sinuses is forced into the middle ear, the accompanying swelling of the eustachian mucous membrane partially or wholly impedes the drainage from the ear, and the conditions are ripe for the development of an infection of the middle ear. My observations in many hundreds of cases of acute purulent otitis media show that in practically 100 per cent there is coexisting acute purulent sinusitis.

Theoretically, it seems possible for the middle ear to become infected from organisms blown up the eustachian tube from the throat or nasopharynx without the occurrence of an accompanying sinusitis, but in such cases the patulence of the eustachian tube is the protective factor, for without the rhinitis and sinusitis present there are not apt to be inflammation and swelling of the eustachian mucous membrane, and therefore the infected material blown into the middle ear is rapidly removed down the tube by mucous secretion and ciliary action before actual infection takes place.

While for years I have felt convinced that the association of acute sinusitis and otitis has been very close, I had no idea that there was such a high percentage of association until recently, when I began to record this association in a consecutive series of cases. This series is

small, consisting of 150 cases of acute otitis media observed in the Children's Hospital of Philadelphia, the University of Pennsylvania Hospital and the St. Christopher's Hospital for Children during the months of February, March and April of this year.

The series consists of consecutive unselected cases, all bed patients in the hospitals, who had been admitted for such conditions as pneumonia, malnutrition, gastro-intestinal disorders and tuberculosis. Other interesting associations of sinusitis or otitis with these various disorders were discovered, but in a paper of this length only the related conditions of sinusitis and otitis will be discussed.

METHOD

The procedure of examination and diagnosis of the sinus infection was as follows: (1) A gross inspection of the nose, the degree of congestion of the nasal mucous membrane, the engorgement of the turbinates, the amount of irregularity of the septum and the amount and character of the secretion being noted; (2) the application of a 2 per cent solution of cocaine hydrochloride to the nasal mucous membrane, especially over the lower turbinate and the under surface of the middle turbinate; (3) introduction of the nasopharyngoscope into each nostril, observation being directed especially to the exact location of pus in the nose; (4) examination of the ears, the character of the discharge (if any) and the condition of the drum and the canal being noted; (5) examination of the throat, with special reference to the condition of the tonsils and the presence or absence of postnasal secretion, and (6) roentgen examination in selected cases.

It is surprising to find with what speed and ease such examinations can be made, even in infants. The average time for the careful examination of a child by the aforementioned method was about eight minutes. In only a few instances was it found impossible to use the nasoscope satisfactorily. At times, a small amount of bleeding occurred, but in no case was this enough to require measures for its control, and in no case was there a deleterous reaction to the slight traumatism, even in extremely sick children. Occasionally, an extremely thick and irregular or deviated septum prevented the insertion of the nasoscope in one or both nostrils, and in a few of the premature infants the nostrils were too small to admit the instrument. However, in most of the smaller infants the nasoscope could be inserted with little or no trouble, and in one premature infant, weighing 3½ pounds (1,587 Gm.) satisfactory examination was made with this instrument.

The use of a 2 per cent cocaine solution in the nose preliminary to the examination may be criticized by some, but in several hundred children, including premature infants, I have not seen the slightest reaction that could be attributed to its use. This has convinced me that the frequently reported reactions that have been attributed to the use of cocaine in the nose have been due not to the action of the drug

Cases of Associated Sinusitis and Otitis in Patients Under 2 Years of Age

No.	Hospital	Sex	Age, Mos.	Condition of Ears	Examination with Nasoscope	Roentgenologic Report
1	C. H.	M	5	Both drums inflamed and bulging; both incised with release of much pus	Small amount of pus in both spheno-ethmoidal recesses	Antrums small and dense
2	C. H.	F	3	Left ear normal, right drum pale, full and bulging	Large amount of mucopus in both nostrils; source indefinite	Both antrums small but dense due to infection
3	C. H.	F	5	Both drums inflamed and bulging; both incised with release of much pus	Small amount of pus in both pheno-ethmoidal recesses, probably	Sinuses poorly developed; antrums very small and dense, apparently due to pus
4	C. H.	M	11	Both drums pale, swollen and slightly bulging	Small amount of mucopus in both middle meatuses	Both antrums and ethmoids dense due to pansinusitis
5	C. H.	M	11	Drums pale, thick and slightly bulging; recent purulent discharge	Small amount of mucopus in right middle meatuses and spheno-ethmoidal recess; could not insert nasoscope in left side—deviation of septum	Marked density in each antral and ethmoidal area due to pansinusitis
6	C. H.	F	3	Right drum pale and bulging; incised, much pus; small amount of pus in left ear	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	Antrums so small, doubt if they could be infected
7	C. H.	F	6	Moderate amount of pus from the left ear; right drum pale and bulging	Large amount of mucus, probably no pus in both nostrils	Both antrums and ethmoids dense, suggestive of infection
8	C. H.	F	6	Right normal; small amount of pus from left; drum thick	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	Marked density in both maxillary and ethmoid cells due to pus
9	C. H.	M	8	Profuse pus from left; none from right; drums pale and swollen	Nose so full of mucopus, examination was unsatisfactory	Right maxillary dense, suggestive of infection
10	C. H.	M	8	Both drums inflamed and bulging; incised with release of much pus; reincised subsequently	Large amount of mucopus in both spheno-ethmoidal recesses	Maxillary sinuses and ethmoids dense due apparently to infection; suggestive of pus
11	U. P.	F	5	Right drum inflamed and bulging; incised with release of pus; left drum pale and slightly bulging; incised—serum	Large amount of pus in left middle meatus and spheno-ethmoidal recesses; none in right	Marked density in region of ethmoid and maxillary sinuses; doubtful whether due to infection or lack of development
12	U. P.	F	5	Both drums inflamed and bulging; incised; release of pus	Large amount of mucus and perhaps some pus in both nostrils, source indefinite	
13	C. H.	M	9	No pus; both drums inflamed and swollen; incised with release of pus	Mucopus in both spheno-ethmoidal recesses	Left ethmoid hazy; may be due to infection
14	C. H.	M	2	Drums pale, thick and slightly bulging; later incised with release of pus	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	
15	C. H.	F	9	Both drums pale and bulging; both incised with release of pus; reincised four times	Dried mucopus in vestibule; no definite pus from sinuses	
16	C. H.	M	7	Recent purulent otitis; both drums dull, red and swollen	Small amount of pus in both spheno-ethmoidal recesses; none in middle meatuses	
17	C. H.	M	3½	Profuse pus from left; none from right; incised; both drums pale and swollen	Large amount of pus in both middle meatuses	
18	C. H.	M	5	Small amount of pus from right; none from left; both drums pale and swollen	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	
19	C. H.	M	5	Moderate amount of pus from both; drums pale and swollen	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	
20	C. H.	M	2	Both drums dull, red and thick; not bulging; recent purulent otitis	Small amount of pus in both spheno-ethmoidal recesses; none in meatuses	
21	C. H.	M	4	Left ear normal, right drum pale, thick and bulging; reincised, pus	Profuse pus in both nostrils; examination with nasoscope unsatisfactory—narrow nostrils	

22	C. H.	M	5	Both drums inflamed and bulging; both incised with release of much pus	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses
23	C. H.	F	5	Both drums slightly inflamed and slightly bulging; incised—thin pus	Small amount of pus in both spheno-ethmoidal recesses
24	C. H.	F	11	Left normal; right drum pale, thick and perhaps slightly bulging	Profuse pus in both middle meatuses; small amount in recesses
25	C. H.	M	9	Both normal; later, bilateral abscesses developed	Small amount of pus in recesses; later, large amount in both middle meatuses and recesses
26	C. H.	F	11	Profuse pus from both.....	Large amount of pus in both middle meatuses; smaller amount in recesses
27	C. H.	F	6	Right normal; left drum pale, swollen and perhaps slightly bulging	Probably some pus in left spheno-ethmoidal recess
28	C. H.	F	5	Both drums inflamed; left swollen and slightly bulging	Probably pus in both spheno-ethmoidal recesses
29	C. H.	F	10	Small amount of pus from right; drum dull, red and thick; left drum bulging; incised pus	Pus in both middle meatuses and spheno-ethmoidal recesses
30	C. H.	M	9	Profuse pus from left; none from right; drum pale and bulging	No actual pus seen, but intranasal appearance suggestive of sinusitis
31	C. H.	M	8	Moderate amount of pus from right; drum dull, red and swollen; left drum inflamed and bulging; incised—pus	No actual pus seen; mucous membrane much inflamed
32	C. H.	M	6½	Both drums pale, thick and full; later both discharged pus	No pus seen, but intranasal appearance suggestive of sinusitis
33	C. H.	F	5	Moderate amount of pus from both; drums pale, swollen and bulging	Large amount of mucopus in both middle meatuses
34	C. H.	F	2 days†	Both drums pale and thickened; not bulging	Mucopus discharged from left nostril; nostrils too small for nasoscope
35	C. H.	M	5	Profuse pus from both; drums dull, red and swollen	Large amount of pus in both spheno-ethmoidal recesses; small amount in meatuses
36	C. H.	M	3	Both drums inflamed and bulging; incised with release of much pus	Unsatisfactory because of markedly deviated septum; pus drawn from both nostrils by suction
37	C. H.	F	6	Both drums pale, thick and slightly bulging	Unsatisfactory because of deviated septum; profuse mucopus from both nostrils
38	C. H.	F	3	Both drums pale, thick and slightly bulging; recent discharge of pus	Large amount of pus in right middle meatus and spheno-ethmoid recesses; left nostril deformed because of operation for hare lip
39	C. H.	M	2	Both drums dull, red, thick and swollen; recent purulent discharge	Small amount of pus in both spheno-ethmoidal recesses; none in meatuses
40	C. H.	M	4	Left normal; right drum bulging; reincised—pus	Unsatisfactory because of very narrow nostrils; profuse pus from both nostrils
41	C. H.	F	3	Both drums inflamed and bulging; incised; pus	Pus in both middle meatuses and both spheno-ethmoidal recesses
42	C. H.	F	8	Both drums inflamed and bulging; incised; much pus	Large amount of mucopus in both middle meatuses and spheno-ethmoidal recesses
43	C. H.	F	11	Left normal; right drum dull, red and swollen; not bulging	Small amount of pus in right spheno-ethmoidal recess; none in left
44	U. P.	M	3½	Profuse pus from right; moderate amount from left; drums thick and bulging	Large amount of pus in right spheno-ethmoidal recess; small amount in left; none in meatuses

† Weight, 3½ pounds (1,586 Gm.); 7½ months premature.

Cases of Associated Sinusitis and Otitis in Patients under 2 Years of Age—Continued

No.	Hospital	Sex	Age, Mos.	Condition of Ears	Examination with Nasoscope	Roentgenologic Report
45	U. P.	M	2	Both drums inflamed, thickened and slightly bulging	Nostrils too narrow for nasoscope; suction drew pus from both	
46	U. P.	F	8	Right normal; left drum dull, red and slightly thickened; recent discharge of pus	No actual pus seen, but evidence of recent sinusitis	
47	U. P.	F	3	Both drums pale, slightly bulging (probably)	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	
48	U. P.	M	7	Both drums dull, red and thick; bilateral mastoidectomy recently	Pus in both middle meatuses and right spheno-ethmoidal recess	
49	St. C.	F	10	Drums inflamed and full; not bulging; catarrh, otitis, later right incised	Small amount of pus in both middle meatuses	
50	St. C.	F	3	Both drums inflamed and bulging; incised—large amount of pus	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	
51	St. C.	M	3½	Both drums inflamed and slightly bulging; incised—pus from left; none from right	Pus in left middle meatus; right nostril too small for nasoscope; weight, 6½ pounds	
52	St. C.	M	5½	Pus discharged from both; bilateral mastoidectomy recently	Pus in both meatuses and probably in spheno-ethmoidal recesses	
53	St. C.	M	4	Profuse discharge of pus from left; none from right; right drum dull, red and thick; later incised—pus	Small amount of pus in both spheno-ethmoidal recesses; none in middle meatuses	
54	St. C.	M	10	Profuse pus from right; left drum inflamed and bulging; incised—much pus	Large amount of pus in both middle meatuses and right spheno-ethmoidal recess	
55	St. C.	F	2	Left normal; moderate amount of pus from right; drum dull, red and thick	Pus in both spheno-ethmoidal recesses and right middle meatus	
56	St. C	F	2½	Left normal; right drum inflamed and bulging; incised—pus	Large amount of pus in right middle meatus; probably also in recess	
57	St. C.	M	4	Both drums inflamed and bulging; incised with release of pus	Large amount of pus in both nostrils; apparently bilateral maxillary and ethmoidal sinusitis	
58	St. C.	M	3	Both drums inflamed and bulging; incised with release of much pus	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	
59	St. C.	F	7	Both drums inflamed and bulging; incised—much pus	Small amount of pus in both spheno-ethmoidal recesses; large retropharyngeal abscess incised	
60	St. C.	M	10	Both drums dull, red and thickened; probably recent purulent otitis	Dried secretions (pus) in both nostrils; no definite pus from sinuses	
61	St. C.	M	4	Both drums dull red and greatly thickened	Large amount of mucopus in right middle meatus and spheno-ethmoidal recess; probably none in left	
62	St. C.	F	7	Both drums inflamed and bulging; incised—pus	Pus in both nostrils; nasoscope indefinite	
63	St. C.	M	4	Moderate amount of pus from right; both drums inflamed and bulging; left incised—pus	Pus in left middle meatus and spheno-ethmoidal recess; deviated septum prevented insertion of nasoscope in right nostril; pus obtained on suction	
64	St. C.	M	6	Both drums dull, red and slightly thickened	Small amount of pus in both spheno-ethmoidal recesses	
65	St. C.	F	9	Profuse pus from left; right drum inflamed and swollen	Mucopus in both middle meatuses	

66	St. C.	M	8	Dried pus in both; drums dull, red and thickened; apparently recent purulent otitis	Small amount of pus in both spheno-ethmoidal recesses; none in meatuses	
67	St. C.	F	7	Both drums inflamed and bulging; incised—pus	Pus in both middle meatuses and both spheno-ethmoidal recesses	
68	St. C.	M	3½	Both drums inflamed and bulging; incised—large amount of pus	Small amount of pus in left middle meatus; none from other sinuses	
69	St. C.	M	11	Right drum inflamed and bulging; incised—pus; left drum slightly inflamed and swollen	Pus in both spheno-ethmoidal recesses	
70	St. C.	M	2	Both drums slightly inflamed and full in appearance; no bulging	Profuse pus from left nostril; deviated septum prevented nasoscope examination	
71	St. C.	M	10	Both drums inflamed and bulging; incised—pus	Mucopus in both middle meatuses and spheno-ethmoidal recesses	
72	C. H.	F	5	Both drums pale and bulging; incised—small amount of pus	Large amount of pus in both spheno-ethmoidal recesses; smaller amount in middle meatuses	
73	St. C.	M	8½	Profuse foul pus from both; drums swollen; not bulging	Moderate amount of pus in both spheno-ethmoidal recesses and middle meatuses	
74	St. C.	M	8	Profuse pus from both; drums much swollen	Large amount of pus in both spheno-ethmoidal recesses and middle meatuses	
75	St. C.	F	7	Both drums inflamed and bulging; incised—pus	Small amount of mucopus in both spheno-ethmoidal recesses	
76	St. C.	M	2	Both drums pale, swollen and bulging; both incised; serum from left—pus from right	Moderate amount of pus in both spheno-ethmoidal recesses	
77	St. C.	M	6½	Profuse discharge of pus from right; left drum inflamed and bulging	Small amount of pus in both spheno-ethmoidal recesses	
78	St. C.	F	2	Both drums inflamed and bulging; both incised—pus	Large amount of pus in both spheno-ethmoidal recesses and middle meatuses	
79	St. C.	M	4	Profuse discharge of pus from both; drums inflamed and swollen	Small amount of mucopus in both spheno-ethmoidal recesses and middle meatuses	
80	C. H.	F	11	Profuse discharge of pus from right; left ear normal	Large amount of pus in both middle meatuses and both spheno-ethmoidal recesses	
81	C. H.	M	9	Both drums inflamed and bulging—incised pus	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	
82	C. H.	F	6	Both drums pale and bulging; recent pus discharged from both	Small amount of pus in both spheno-ethmoidal recesses and right middle meatuses	
83	C. H.	M	7½	Right drum dull, red, swollen and bulging; left slightly swollen; recent discharge from both	Small amount of pus in both middle meatuses	
84	C. H.	M	3½	Both drums inflamed and bulging; incised—pus	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	
85	C. H.	M	5	Recent purulent otitis; drums dull, red and swollen	Pus in both middle meatuses and spheno-ethmoidal recesses	
86	C. H.	M	17 days‡	Both drums pale, swollen and bulging.....	No definite pus but large amount of mucus	
87	C. H.	M	23	Profuse pus from both; drums red and swollen	Small amount of pus in both spheno-ethmoidal recesses	
88	C. H.	M	12	Moderate amount of pus in both; drums pale and bulging; both drums reincised several times	Large amount of pus in both meatuses; small amount in both spheno-ethmoidal recesses	
89	C. H.	M	13	Profuse pus from right; none from left; both drums swollen, dull and red	Profuse mucopus in both middle meatuses and spheno-ethmoidal recesses	
90	C. H.	M	19	Both drums inflamed and bulging; both incised—release of pus	Small amount of pus in both middle meatuses and both spheno-ethmoidal recesses	Increased density in right antrum, suggesting pus

‡ Weight, 4½ pounds (2,040 Gm.)

Cases of Associated Sinusitis and Otitis in Patients Under 2 Years of Age—Continued

No.	Hospital	Sex	Age, Mos.	Condition of Ears	Examination with Nasoscope	Roentgenologic Report
91	C. H.	M	14	Both drums slightly inflamed and bulging	Pus in both middle meatuses	
92	C. H.	M	14	Both drums dull, red and thick, slight bulging	Pus in left spheno-ethmoidal recess; could not insert nasoscope in right—deviated septum	
93	C. H.	F	18	Both drums slightly inflamed and full, not bulging; catarrhal otitis	Small amount of pus in both middle meatuses and spheno-ethmoidal recesses	
94	C. H.	F	15	Left drum inflamed and bulging; incised; pus; right drum slightly inflamed and thick	Large amount of mucopus in both middle meatuses and spheno-ethmoidal recesses	
95	C. H.	M	14	Both drums slightly inflamed and swollen; not bulging	No evidence of sinus infection in right nostril; could not insert nasoscope in left nostril (deviated septum)	Increased density in right antrum; left not entirely clear; suggested pus
96	C. H.	M	18	Both drums dull, red, thick and slightly bulging; incised—much pus	Mucopus in both spheno-ethmoidal recesses and right middle meatus	
97	C. H.	F	19	Left drum dull, red and swollen; right drum inflamed and bulging; incised—pus	Large amount of pus in both nostrils; source indefinite	
98	C. H.	M	14	Both drums dull, red, swollen and slightly bulging	Large amount of pus in both spheno-ethmoidal recesses and middle meatuses	
99	C. H.	F	18	Right drum inflamed and bulging; incised—pus; left normal	Small amount of pus in both middle meatuses	
100	St. C.	F	20	Both drums inflamed and bulging; incised—much pus	Large amount of pus in both spheno-ethmoidal recesses; smaller amount in meatuses	
101	St. C.	F	20	Both drums dull, red and thickened; recent purulent otitis	Mucopus in both nostrils; nasoscopic observations indefinite	
102	St. C.	M	17	Both drums pale and bulging; incised—small amount of pus	Mucopus in both middle meatuses, probably also in recesses	
103	St. C.	M	20	Right normal; left drum inflamed and bulging; incised—pus; both drums incised 18 days later	Large amount of mucopus in nasopharynx; nasoscopic findings indefinite	
104	St. C.	M	12	Profuse thick pus from both; drums dull, red and swollen	Small amount of pus from both spheno-ethmoidal recesses	
105	St. C.	M	20	Left drum inflamed and bulging; incised; right normal, later incised	Small amount of mucopus in both spheno-ethmoidal recesses	
106	St. C.	F	19	Both drums dull, red and thickened; recent otitis media	Small amount of mucopus in both spheno-ethmoidal recesses and left middle meatus	
107	St. C.	M	18	Moderate amount of thick pus from right drum; much swollen; left normal	Large amount of pus in both middle meatuses and spheno-ethmoidal recesses	
108	St. C.	F	18	Left normal; right drum pale and bulging; incised—pus	Large amount of pus in both spheno-ethmoidal recesses	
109	C. H.	M	13	Profuse foul discharge of pus from both; chronic condition	No pus visible; none obtained by suction	
110	C. H.	M	15	Right normal; left drum inflamed and thickened; not bulging	Pus in left middle meatus; none in right	

* C. H. indicates the Children's Hospital of Philadelphia; U. P., the University of Pennsylvania Hospital, and St. C., St. Christopher's Hospital for Children.

(except when used in excessive amounts), but probably to the psychic disturbances that so often accompany instrumentation in the nose, particularly in young adults.

Of course, the amount of cocaine solution used in these patients has been very small. A rather tightly wound cotton applicator is dipped into the 2 per cent solution, the excess pressed out and the applicator then rubbed along the floor of the nose, the lower turbinate and the under surface of the middle turbinate. A minute and a half or two minutes of rubbing suffices for excellent anesthesia and sufficient constriction of the congested nasal vessels and turbinates for satisfactory examination.

The use of the nasoscope in the diagnosis of sinusitis is easy and satisfactory, more so than the use of roentgenograms. In many cases the nasoscope is not necessary for diagnosis, the profuse purulent secretion from a nostril indicating a purulent sinusitis. Frequently, it is necessary to remove some of the purulent and excessive mucus secretion before a satisfactory view with the nasoscope can be obtained. For this purpose I find the best instrument to be a hand suction bulb containing a glass tip to fit the nostril. Such an instrument, if used properly, not only removes the secretions lying in the nostrils but also draws the pus from the sinuses. In some of the cases pus could not be seen with the nasoscope at first, but after the use of suction it could be observed near the maxillary orifice or in the ethmoid region. When the nose is sufficiently clear, the nasoscope is inserted and the location of pus determined. From a diagnostic standpoint, it is satisfying to see with the light pus lying well up in the middle meatus or sphenoethmoidal recess or exuding in a small stream from the maxillary orifice.

When pus is observed in the middle meatus the conclusion can be reached that it is coming from the anterior ethmoidal cells, the maxillary sinus, or both. When a large amount of pus is present, I have assumed that both sinuses are involved. At times the nasoscope will show pus coming from the maxillary orifice in a stream and the presence of more pus overlying the ethmoidal cells indicating infection of both sinuses. When the pus is seen in the spheno-ethmoidal recess, it indicates infection of the posterior ethmoidal cells, chiefly, but probably also at times infection of the sphenoid, especially in older children. These assumptions have been confirmed by roentgenograms sufficiently often to be established as facts.

In this series of cases, roentgenograms were made chiefly in those cases in which the nasoscopic observations made the existence of sinus infection doubtful and, in some instances, to corroborate the clinical findings. Examination with the nasoscope was found to be much more satisfactory for diagnosis than the taking of roentgenograms, especially in the smaller children. Reports would often state that the obscurity

of the sinuses in the films was due either to undeveloped sinuses or to infection. At other times the report would be uncertain because of movements of the child.

I wish to take exception to the statement that roentgenograms are indispensable in making an accurate diagnosis of sinusitis in children. To my mind nothing could be more accurate or satisfactory than actually to see the pus coming from the maxillary orifice or lying in the sphenoethmoidal recess. Five minutes' work with the suction bulb and the nasoscope is all that is necessary for accurate diagnosis of sinusitis in infants and children.

Without a satisfactory roentgenogram there may be some doubt concerning which sinuses are involved, especially in older children. In the great majority of cases, however, this is immaterial because in the treatment other than conservative measures are seldom necessary. The essential point is to determine whether sinusitis is present; whether the maxillary or the ethmoid sinuses are involved is of remote importance in the majority of cases.

A factor that is of considerable interest in the study of such sinus infections in children is the frequency of involvement of the middle ear. Every otolaryngologist in a children's hospital is sooner or later confronted by the necessity of controlling so far as possible the widespread prevalence of acute otitis media among the infant patients. It is discouraging to the pediatrician treating an infant with a gastro-intestinal or a feeding disorder in the hospital to have otitis media suddenly develop in his patient. Nurses at times feel that it is a reflection on their ability when such a condition develops in a child under their care. What factors are present that account for the otitis media, and what can be done to control it?

The causal conditions present are evident. A study of them convinces one that otitis media seldom occurs without an accompanying cold, which practically always means sinusitis. The infected material from the sinuses is coughed, sneezed or vomited up the short, wide and patulous eustachian tube and infects the middle ear. When one considers the frequency of such colds or sinus infections in infants and children, it is not surprising that otitis media occurs so often, but that it does not occur more frequently, for it seems impossible to prevent the acute rhinitis. The children are in the hospital for ailments that would tend to lower their resistance to infections; most of them have such conditions as dietary deficiency, allergic reactions, endocrine disorders and metabolic disturbances, which make them more susceptible to infections of the mucous membrane. In addition to these factors, the active infective agent is ever present, in the patient themselves, in

the visiting physicians or in the attending nurses. It does not seem possible to isolate a child, especially in a hospital, so thoroughly that the infecting organisms cannot reach him.

In an attempt to prevent infections of the ear every possible precaution should be observed in connection with the transmission of infecting organisms, the elimination so far as possible of underlying conditions, and, what I believe is more practical, the early recognition and treatment of sinus infections.

Both as a means of preventing aural infections and as a therapeutic measure for sinusitis, I believe that the most practical treatment is the suction treatment of the nose for removal of the secretions. In one of the children's hospitals where I am in attendance, I have had suction bulbs placed in every ward and instructed the nurses in their use. When colds develop, the bulbs are used frequently during the day to remove nasal secretions. During the past winter I have observed in patients so treated a noticeable reduction in the number of abscesses of the middle ear and a more rapid recovery from sinusitis. The use of organic silver preparations in the nose appears to me to be useless. Unless the secretions are thoroughly removed before instillation of a mild silver protein solution the drug does not come in contact with the nasal mucous membrane to any effective extent, and it only causes further blocking of nasal respiration and is a source of irritation to the mucous membrane. There may be some antiseptic value in this as well as in other commonly used nasal preparations; but of what use is it to kill the bacteria in these secretions? The organisms have already done their harm and are on their way out of the system with the secretions, and the logical treatment would seem to be to remove them as fast as possible. If such antiseptics could be applied directly to the infected sinuses, which are the source of most of these secretions, they could be of some value; even so, their use would still be questionable, because most antiseptics kill only the bacteria on the surface of the mucous membrane and have no penetrating effect. It is the infection in the mucosa, submucosa and deeper layers of the lining membrane of the sinuses that does the harm and not the bacteria on the surface and in the secretions. Kistner [2] and Watson-Williams [3] have satisfactorily proved the existence of the infecting organisms in the tissues lining the sinuses in both acute and chronic infections of the sinuses. Therefore, unless the antiseptic can penetrate the mucosa and kill the bacteria there, it can be of little or no benefit. The rational

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2. Kistner, F. B.: Histopathology and Bacteriology of Sinusitis, Arch. Otolaryng. **13**:2 (Feb.) 1931.

3. Watson-Williams, P.: Remarks on Tissue Invasion of Low-Grade Pyogenic Organisms, J. Laryng. & Otol. **44**:598, 1929.

treatment in acute sinus infections in children appears to me to be the removal of the secretions by some type of suction, rather than by an attempt at antisepsis.

In advocating the use of suction for infections of the sinuses, I am well aware of the difference of opinion prevalent regarding both the efficacy and the dangers of such a procedure. As regards its efficacy, there can be no doubt that suction properly used is of great help in treatment, especially in infants and children. The mucopurulent secretions can readily be withdrawn by the hand suction bulb, not only from the nasal chambers but also from the sinuses. In children the ostia of the sinuses are large in proportion to the size of the sinuses, and the secretions are rather easily removed by negative pressure properly used. The tip of the suction bulb must fit tightly in the nostril, and on release of the compressed rubber bulb the air must be allowed to enter the other nostril slowly unless a sufficient amount of air is coming through the nasopharynx. This causes a rather forceful stream of air through the nostril, which removes the secretion in that chamber; by a negative pressure effect and by the cohesive action of the mucopus this material is drawn in large measure from the affected sinuses. Such suction not only removes the nasal secretions but also to some extent tends to withdraw secretions from the infected middle ear through the eustachian tube. I have proved this by the instillation of colored fluid in the aural canal in cases in which perforations in the drum were visible, and after suction with the nasoscope, I have observed this colored fluid in the nasopharynx.

Regarding the possible dangers of negative pressure, I can conceive of harm being done by too forceful suction. However, if air is allowed to slip through the opposite nostril if it is not drawn sufficiently through the nasopharynx, I can see no harm in its use. The rapid subsidence of several cases of orbital cellulitis secondary to ethmoiditis resulting from the use of suction has convinced me of the value of this procedure.

There appears to be a difference of opinion among writers on sinusitis in children as to which sinuses are more frequently infected. Some claim that the maxillary sinus is more often involved, while others (perhaps the greater number) believe that the ethmoid cells are the first in frequency. The cases studied in this series are too few to give an accurate indication of the frequency of involvement of the individual sinuses, but the results appear to favor the contention that the ethmoids are more often infected. When pus was observed in the spheno-ethmoidal recess, a diagnosis of ethmoiditis was made, and when it was found in the middle meatus, a diagnosis of anterior ethmoiditis or maxillary sinusitis, or both, was made. Occasionally, pus could be seen coming from the maxillary orifice; then an accurate diagnosis of maxillary infection could be established. Pus was found in the spheno-

ethmoidal recess more often than in the middle meatus, indicating a more frequent involvement of the ethmoidal sinuses. However, in the great majority of cases, pus was found not only in both regions, indicating infection of both groups of sinuses, but also on both sides, showing bilateral involvement. This study further revealed that it was more common for all the sinus spaces to be involved in infants than in older children, the latter often showing infection of only one sinus.

An interesting feature occasionally observed was the presence of pus in one nostril while the ear on that side was normal and otitis media was present in the other ear. When such cases were followed, however, infection of the homolateral ear was found later.

In this series, 150 cases of otitis media in children between the ages of 2 days and 11 years were studied. Eighty-five of the children were under 1 year of age and 110, under 2 years. In 140 of the 150 cases, or 93.3 per cent, a positive diagnosis of purulent sinusitis was made by means of the nasoscope. Of the remaining 10 cases, six were classified as doubtful, because no pus could be seen but the intranasal appearance indicated probable infection of the sinuses. Only 4 patients showed no evidence of sinus infection when examined by the nasoscope, and in each of these, a thick purulent secretion had been draining from the ears for long periods, suggesting that the otitis was of the chronic type. In such cases, the purulent sinusitis probably present at the start of the otitis had apparently cleared up. If the 6 doubtful but probable cases of sinusitis are classed with the positive ones, there is a total of 97.3 per cent of cases in which acute sinusitis accompanied the otitis. In only 25 cases were roentgenograms of the sinuses made, and in only a single case in which there was nasoscopic evidence of sinusitis were the roentgen findings negative. The patient was an infant, 3 months of age, who showed a large amount of pus in both middle meatuses and both spheno-ethmoidal recesses. The report from the roentgenologist stated that the antrums were so small that it was doubtful that they could be infected. No mention was made of the ethmoids.

Of the 85 infants under 1 year with otitis media, definite infection of the sinuses was found in 97.5 per cent and probable infection in 100 per cent. While the total number of cases is small, it represents a consecutive unselected group and is therefore probably an accurate indication of the percentage of association of the two conditions. Therefore, it seems reasonable to conclude that when an aural discharge is present in an infant, sinusitis is also present. This should be of considerable significance to the pediatricians, who for the past few years have stressed the importance of infection of the ear as a factor in gastro-intestinal disturbances of infants but who have not taken into consideration the coexisting sinus infection. If, as they assume, infections of the ears have such a deleterious influence on the infant, how

much greater effect would the infected sinuses have, which produce perhaps twice as much pus as do the ears. It seems to me that infected sinuses are of more importance than infected ears in gastro-intestinal conditions, and that it would be as rational for the pediatrician to advocate operative treatment for such conditions of the sinuses as to insist that mastoidectomy be performed. My plea is for conservative treatment of such conditions of the ear by active treatment of the coexisting sinus infections, for improvement in the ears closely follows or precedes the improvement of the sinus infection.

One fact clearly demonstrated in this study of sinusitis in children is that the younger the child with sinusitis, the more likelihood is there of aural complications. In children under 1 year of age the association of sinusitis and otitis was common. In the examination of many older children with purulent sinus infection, the ears were found to be involved in a far smaller percentage of cases. This should be expected, for the older children are more able to remove the nasal secretions, and the eustachian tubes are gradually assuming the adult type.

In some of the older children purulent otitis media was found without an associated sinus infection. Some of these infections of the ear were of the chronic type, the discharge from the ear persisting after the sinus infection had subsided. On the whole, however, the aural infection appeared to parallel the sinus infection. As the discharge from the nose diminished, the aural infection subsided, and the condition of the ear often cleared up while there was still considerable secretion from the sinuses. In other cases sinusitis was found in a child whose ears were normal. In such instances, especially in infants, it could be prophesied with considerable accuracy that the ears would later become infected. Usually within a few days both ears would become involved.

In the examination of the ears particular attention was given to the study of the drum and the variations in its appearance with the different conditions present in the nose and the sinuses. In the presence of acute purulent sinusitis, the drum was found in several degrees of involvement, ranging from a normal condition to typical purulent otitis. The factors controlling the degree of involvement of the middle ear in association with sinusitis are of great interest and are concerned with the virulence of the infection, the resistance of the patient, the patulence of the eustachian tube and the amount of unremoved secretions. In some cases, the virulence of the infection or the lack of resistance of the patient may be so marked that mere contamination of the tympanic mucosa is followed by rapid progress of the otitis media. This gives the textbook picture of a severely inflamed and swollen drum, rapid accumulation of pus and rupture of the membrane within a short time. This is a common picture in cases of otitis, but it is probably

not so common as in the cases in which the drum has an entirely different appearance. For instance, in catarrhal otitis, which is so often mistaken for the infected purulent type, the drum appears rather full, owing to the transudated serum, but there is little or no congestion. Drums in this condition are often wrongly incised at the insistence of the pediatrician, who perhaps does not take into consideration the noninfective nature of this type of otitis. Again, there is the pale, swollen edematous drum, which may bulge somewhat from pus in the tympanic cavity owing to drainage of the unremoved secretions from the nose and nasopharynx. Owing, perhaps, to a low virulence of the infecting organism, such a drum may show no congestion, and the tympanic cavity may act only as a reservoir for excessive secretions; if the eustachian tube remains patulent or semipatulent, such secretions may readily discharge back into the nasopharynx. Such conditions may not require myringotomy or active treatment of the ear, since drainage through a myringotomy opening does not occur. An incision is made and pus is released, but the next day the discharge has ceased, and the drum is again bulging. I have incised such a drum numerous times but have failed to establish continuous drainage, and it is my opinion that such a condition does not require myringotomy, or at least, not a second myringotomy. When the ear does not drain after an incision of the drum, it means that the tension of pus in the middle ear is not sufficient to force pus out of the opening and to keep the incision open. There is not enough tension to do this because the pus is draining down the eustachian tube as fast as it accumulates. Therefore one should not continue to incise such a drum, for every such incision means more scar tissue, fibrous tissue formation and, perhaps, adhesions in the healing process. Furthermore, it is quite possible that the pus may be entirely sterile. Ashley and Frick [4] have shown that pus may be irrigated from the maxillary sinus and found to be sterile on culture. Such pus may find its way into the middle ear and produce an appearance such as that just described. It was interesting to note in this series of cases that such an appearance of the drum was usually accompanied by mucopurulent secretion rather than by real pus in the nose, as seen by the nasoscope.

Another type of involvement of the middle ear must be distinguished, the type that occasionally accompanies allergic nasal reactions. Such a condition may cause an excessive secretion of mucous in the nose and nasopharynx, which, in the case of infants, may be blown into the middle ear and produce the fulness of the drum so

4. Ashley, B. J., and Frick, B. S.: A Bacteriologic and Cytologic Study of the Maxillary Antrum in Children, Ann. Otol., Rhin. & Laryng. **39**:605 (June) 1930.

commonly seen in nasal irritations. Incision of the drum in such cases often results in the outflow of a secretion which is practically all mucus and probably of a noninfectious character.

It appears to me that in the past the treatment of many conditions of the ear has been too radical. This is at times due to the insistence of the pediatrician, who is anxious to have eliminated all underlying conditions that may have a bearing on the condition for which the child is being treated. The primary myringotomy is often not necessary, and the frequent subsequent incisions are seldom needed. The proper handling of such cases is the active treatment of the associated sinus infection and the maintenance of drainage of the middle ear through the eustachian tube. Of course, in the virulent infections with accompanying severe inflammation, active treatment is needed, but in such cases, reincision of the drum is seldom necessary, for the rapid formation and discharge of pus tend to keep the incision open.

CONCLUSION

A report of a rather small number of cases in which acute sinusitis was found in practically 100 per cent of the cases of acute otitis media does not necessarily prove that all cases of otitis are secondary to sinusitis, but it at least suggests the common association of the two conditions. Furthermore, it suggests that in cases of otitis media the treatment should not be confined to the ear, but should be largely directed to the elimination of the sinus infection. This can probably be done best by the frequent and thorough removal of the secretions, little stress being placed on the use of antiseptics. The results of this study further emphasize the importance of considering more seriously infection of the sinuses as a contributing factor in gastro-intestinal conditions as well as many other disorders of infancy. It is to be hoped that with the realization of the frequency of sinus infection in infants and children and the direct and remote complications which ensue therefrom more adequate prophylactic and therapeutic measures may be discovered to combat such conditions.

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ABSCESS OF THE PTERYGOMAXILLARY FOSSA COMPLICATING OTITIC INFECTIONS

REVIEW OF THE LITERATURE AND REPORT OF A CASE

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There seems to be a scarcity of references in the literature on abscesses of the pterygomaxillary or infratemporal fossa. In this paper I therefore wish to review the literature and to report a case.

The pterygomaxillary, infratemporal or zygomatic fossa is the space bounded laterally by the ramus of the mandible, medially by the lateral wall of the pharynx and pterygoid process of the sphenoid bone, anteriorly by the zygomatic surface of the superior maxilla and superiorly by the lower surface of the greater wing of the sphenoid and the adjacent temporal bone. The posterior limit of the space is represented by a plane passing directly medialward from the posterior border of the ramus of the mandible to the pharynx.

The contents are the internal and external pterygoid muscles, the internal maxillary artery with its branches and companion veins, the pterygoid plexus of veins, the inferior maxillary nerve and its branches, the chorda tympana nerve, a part of the parotid gland, internal maxillary lymph glands and the internal lateral ligament of the mandible.

This description is the one given in Deaver's "Surgical Anatomy," most other works on anatomy describing it as the zygomatic fossa.

In 1908, Foster [1] reported a case of abscess in the zygomatic fossa in a woman, 55 years of age, who awoke in the morning with pain in the left cheek and eye, with some swelling of the side of the face and congestion of the eyeball, having retired the evening before apparently well. The gum around a carious tooth was incised, but no pus was found. Owing to the patient's inability to open the mouth, the physician in charge of the case stated that he was unable to make the incision far enough back in the mouth. This in itself should have been a valuable clue to the diagnosis. After a few days, a profuse flow of pus appeared from the incision, but the swelling of the face and the exophthalmos became great in spite of the discharge. Twenty-six days after the onset, the exophthalmos and swelling of the side of the face were intense, and an incision was made through the conjunctiva in the

1. Foster, M. L.: Abscess in the Zygomatic Fossa, Ann. Ophth. **17**:439, 1908.

lower fornix. No pus was discovered that day, but the next day there was a free discharge from the wound. No diminution of the exophthalmos took place, and exploration of the orbit and antrum was made without pus being discovered. The wound continued to drain for some time, with loss of vision of the left eye. Under ether an incision was made along the lower lid, and the periosteum was elevated until an opening recognized as the sphenomaxillary fissure was brought into view; the bone was in healthy condition, but when a probe was passed through the fissure into the cavity beyond, granulations could be felt. After these granulations had been removed, a pyramidal cavity was found. This was identified as the zygomatic fossa. The condition promptly cleared up after this operation.

The diagnostic points given by Foster in this case are the sudden onset of pain, tenderness and swelling over the side of the face, congestion of the conjunctiva, immobility of the jaw and exophthalmos which was probably caused by a direct extension of the inflammation to cause a retrobulbar cellulitis by the passage of the pus through the sphenomaxillary fissure.

Foster believes that the only way by which this abscess could have been produced was metastasis through the lymph channels from carious teeth in the upper jaw. In his case there was no infection of the ear.

Beck [2] stated that he had observed an unusual case of extradural abscess in the temporosphenoidal region in which necrosis of the thin squamous portion of the temporal bone took place, with the subsequent burrowing of the pus under the temporal muscle and zygoma.

In his report of a case complicating otitic infection, Strauss [3] stated that in a search through the literature he was unable to find a record of the involvement of the pterygomaxillary fossa in connection with otitis media or suppurative mastoiditis, and that indeed mention of the locality from a practical or clinical standpoint was extremely rare. His case was that of a boy, 8 years of age, in whom otitis media developed, paracentesis being necessary within the first twenty-four hours. The discharge gradually decreased until the second week, when it became more profuse, with a rise in temperature and tenderness over the mastoid. The patient complained of a decided pain over the left side of the face which was exaggerated by deglutition, the pain increasing day by day and the patient screaming with agony when swallowing. Strauss stated that there was no swelling in the temporal region or preaural regions. There was a decided limitation of movement of the mandible, the

2. Beck, Joseph: Applied Pathology in Diseases of the Throat, St. Louis, C. V. Mosby Company, 1923, p. 90.

3. Strauss, J. F.: Pterygomaxillary Abscess Complicating Acute Mastoiditis. Ann. Otol., Rhin. & Laryng. **36**:477 (June) 1922.

mouth admitting only the tip of a finger. The tenderness over the mastoid increased; the discharge from the ear increased, and there was a sagging of the posterior superior wall of the canal. A diagnosis of surgical mastoiditis was made and operation was advised. Dr. George Shambaugh concurred in this diagnosis the following day. A roentgenogram showed a pneumatic mastoid which was definitely cloudy. The osseous trabeculae were apparently not broken down. The leukocytes numbered 13,400. A culture of the blood gave negative results. A culture of the aural discharge showed a hemolytic streptococcus. On operation pus was found in all cells, and several areas of softening of the bone were found. The zygomatic root was explored, but it was normal. Following the operation the patient was in no way improved, the facial pain becoming more acute. Morphine gave very brief relief. The patient was constantly shrieking with pain. The symptoms continued for two weeks, during which swelling appeared in the right side of the face, with a gradual increase of temperature and leukocytosis. A definite subdermal fluctuation was seen over the junction of the middle and posterior thirds of the zygoma. While the anesthetic was being given in preparation for an incision, this fluctuation ruptured into the mastoid wound with a reduction of the swelling, and the patient showed marked improvement. Within five days, however, all of the symptoms recurred. Finally, a crucial incision was made into the superficial portion of the abscess and a drain was inserted, resulting in recovery. The opinion of Strauss was that the path of infection was a hematogenous one, that it was caused by diseased or ruptured cells in the digastric groove of the mastoid process or that it was the same mechanism and pathologic process that occur in the cells occasionally found invading the petrous portions of the temporal bone.

McKenzie [4] reported the case of a boy, aged 8 years, admitted to the hospital with a chronic suppuration of the left middle ear with a temperature of 103 F. A radical mastoid operation was performed immediately, with no relief of temperature. Two days later the wound was reopened; a small drop of pus on the floor of the external meatus was found to lead to an abscess in front of the bony meatus. This was opened and drained. Thrombosis of the cavernous sinus and death followed. Autopsy showed an erosion of the outer wall of the jugular bulb leading to the site of the abscess in the pterygomaxillary fossa. There was no evidence of disease of the bone adjacent to the abscess. Dr. McKenzie thinks that this was evidently a case of thrombosis of the jugular bulb which extended down the vein into the neck. The

4. McKenzie, D.: Otitic Pterygomaxillary Abscess Induced by Thrombophlebitis of the Jugular Bulb, Proc. Roy. Soc. Med. (Sect. Laryng.) **16**:53 (Aug.) 1923.

thrombosis had broken down, and the pus had made its way through the wall of the bulb into the pterygomaxillary fossa.

In a search through the "Index Medicus" and the "Index Catalog of the Surgeon General's Library," the cases of abscess of the pterygomaxillary fossa just reviewed are the only ones found recorded. However, I wish to cite a few reports of cases of retropharyngeal abscess complicating otitis media and mastoiditis, as the pathway of infection may be the same.

Pearlman [5] stated that of sixty-one patients with retropharyngeal abscess admitted to the Sarah Morris Hospital in a five year period, nine had otitis media, in seven of whom it was on the same side as the abscess. He stated that the intracranial route is a rare source of infection, the direct source being more common. He stated that the pus may escape by way of the floor of the antrum or through the anterior bony auditory canal wall, with possible involvement of the temporomandibular joint, or it may enter the posterior fossa through the posterior wall of the antrum and hence downward. It may pass along vessel canals by means of bone destruction. He believes that the semicanalis musculis tensoris tympana plays a rôle as a pathway.

Schoeneman [6] reported a case in a man, 35 years of age, who had chronic suppuration of the middle ear and mastoiditis with a periodic discharge through a sinus above the tonsil.

Cunningham,[7] in discussing retropharyngeal abscess complicating otitic infections, considers the lymphatics as one of the routes by which infection from the ear may pass into the pharynx. He gives five routes in which an otogenic retropharyngeal abscess may occur: (1) by infection of a retropharyngeal lymph node; (2) by direct extension from a necrotic area in the mastoid process; (3) by direct extension forward from an infection of the middle ear (he does not say how); (4) by the exit of an extradural abscess from the cranial cavity, and (5) by the secondary burrowing of a Bezold's abscess.

McKenzie [8] stated that the infection of the retropharyngeal lymph nodes with suppuration of the ear is not uncommon in infants, and that even in adults unilateral swelling of the posterior pharyngeal wall is sometimes seen. He stated that an otogenic abscess may arise in the pharynx by direct extension through the temporal bone, by way of the foramen lacerum, and by direct extension from the pneumatic cells in the mastoid to the under surface of the occiput and thence to the

5. Pearlman, S. J.: Retropharyngeal Abscess of Otogenic Origin, Ann. Otol., Rhin. & Laryng. **39**:249 (Jan.) 1930.

6. Schoeneman: Verhandl. d. deutsch. otol. Gesellsch. **18**:307, 1909.

7. Cunningham, H. M.: Tr. Am. Laryng., Rhin. & Otol. Soc., 1927, p. 542.

8. McKenzie: J. Laryng. & Otol. **30**:12, 1915.

pharynx. Indirectly, the process may arise by the wandering of an extradural abscess of the posterior cranial fossa to the suboccipital region.

Piersol [9] stated that the parotid lymph nodes drain the cavity of the middle ear, and that the parotid lymph nodes connect with the internal maxillary lymph glands situated in the pterygomaxillary fossa, which would explain the lymphatic pathway of infection.

There is one other pathway by which I believe infection could pass from the middle ear to the pterygomaxillary fossa, which I have not found mentioned in the literature: The chorda tympana nerve passes from the cavum tympanum to the pterygomaxillary fossa by way of the fissure petrotympanica or glaserian fissure. Wendell Phillips [10] and Piersol stated that the osseous portion of the auditory canal is not present at birth but is represented by a partially formed bony ring, the annulus tympanicus. In the development of the osseous canal a dehiscence filled with fibrous tissue remains between the lower periphery of the ring and the bony ring which forms the outer section of the anterior and lower wall of the meatus. This dehiscence usually becomes filled by bone at the third year, but occasionally bony union never becomes complete. It is this union that forms the glaserian fissure. It therefore seems logical that infection could pass into the pterygomaxillary fossa by this route.

REPORT OF CASE

A. K., a man, aged 38, came to my office on May 7, 1927, stating that he had had a severe pain in the right ear since 3 a. m. The membrana tympana showed a slight injection of the manubrium and loss of luster, with no bulging. There was no tenderness over the mastoid and no temperature. The patient was given a 5 per cent solution of phenol in glycerin and was told to return the next day if there was no improvement or if the condition was worse. The following day, the temperature was 102 F., and there were considerable injection and bulging of the posterior inferior segment of the membrana tympana. Paracentesis was performed, and the patient made a good recovery, with the membrana tympana healing by May 19. Two days later, the patient returned, complaining of a slight pain and a feeling of fulness in the right ear. There was no temperature, and there was a slight injection of the drum with a marked bulging in the region of the old scar. This was incised and was followed by a slight discharge of serum and relief from pain. On May 23, the perforation was healed and the color and landmarks practically normal. On June 6, two weeks later, the patient returned to the office complaining of a severe pain in the region of the right zygoma, which was aggravated by movement of the jaws, and a slight pain on swallowing. He stated that this pain had begun two days previously and had gradually become more severe until now it was becoming unbearable. There was a slight swelling just above and

9. Piersol: Anatomy, Philadelphia, J. B. Lippincott Company, 1920, p. 955.

10. Phillips, Wendell: Diseases of Ear, Nose and Throat, ed. 7, Philadelphia, F. A. Davis Company, 1920, p. 135.

below the zygoma which was slightly tender to pressure; there was no redness or sense of fluctuation, and the drum was normal except for a slight injection of the manubrium. The temperature was normal. Two days later the swelling had increased so that there was a definite depression in the region of the zygoma; there was no redness or induration. The temperature was still normal. The patient could not open his mouth more than an inch because of what seemed to be a rigidity of the muscles, and also because of the pain, which was especially severe on movement of the mandible and on swallowing. A distinct swelling was seen on the posterior wall of the pharynx on the right side, but no fluctuation could be elicited. The drum was slightly injected; there was some bulging, and fluid could be seen in the cavity of the middle ear on a level with the umbo. A paracentesis was done, followed by a discharge of clear serum. During the next three days the swelling continued to increase, mostly above the zygoma. There was no pitting on pressure or fluctuation at any time. The pain was intense and was not relieved by morphine; the patient was unable to open his mouth, and there were difficulty and severe pain on swallowing. The temperature was 99 F., the pulse, 112. The red cells numbered 4,521,000, the leukocytes, 16,000, with polymorphonuclears 64 per cent. The roentgenogram of the mastoid and zygoma showed negative results. On June 10, six days after the beginning of swelling, an incision was made along the upper margin of the zygoma. The fibers of the temporal muscle were separated and on dissection downward under the zygoma over the temporal crest pus began to well up. A grooved directer was passed downward about 2 inches from the zygoma into an abscess cavity in the pterygomaxillary fossa. A soft rubber drain was placed in this cavity. There was little drainage, and the next day the patient had obtained almost complete relief from the pain and difficulty on swallowing. The drain was removed in four days, and the wound healed in a week.

EARLY DIAGNOSIS IN MENINGITIS

CHEMICAL STUDIES OF THE SPINAL FLUID

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Progress in the successful management of otitic meningeal infections is dependent on very early diagnosis. The finding of the bacterial invaders in the cerebrospinal fluid comes too late to permit diagnosis at the stage of the invasion when therapy is effective. The inflammatory reaction produced by various causal agents brought to the parts through divers channels effects characteristic tissue reactions.

One of us (Dr. Kopetzky [1]), in a monograph published in 1912, called attention to the value of establishing early diagnosis in meningeal infections through an estimation of the chemical contents of the cerebrospinal fluid, and alluded to the significance of the abnormal absence of chemical elements normally present and to the presence of an excess of toxic substances and the products of brain metabolism, impeded in their removal from the cerebrospinal system by factors of intracranial pressure and stasis of the cerebrospinal fluid system.

Experience has since taught that even when the factors stressed in that article are studied in a given case, diagnosis is arrived at too late for effective therapy. It must be borne in mind that considerable progress has been made in impeding the advance toward the meninges of otitic infections. Newer pathways [2] have been located, and the penultimate clinical picture [3] developed which intervenes between the otitic infection and the terminal lesion in the meninges. Believing that the delicate interchange of ionic constituents which takes place physiologically when disturbed by the commencement of an infection would show abnormal reaction and that these reactions, when accorded their proper significance, might give additional diagnostic data, we under-

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1. Kopetzky, Samuel J.: Meningitis: Nature, Cause and Principles of Surgical Relief, Tr. Am. Laryng., Rhin. & Otol. Soc. **18**:116, 1912.

2. Eagleton, Wells P.: Unlocking of the Petrosal Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex, Arch. Otolaryng. **13**:386 (March) 1931.

3. Kopetzky, S. J., and Almour, R.: The Suppuration of the Petrous Pyramid. Ann. Otol., Rhin. & Laryng. **39**:996, 1930; **40**:157, 396 and 922, 1931.

took a series of chemical examinations of cerebrospinal fluid taken from patients with meningeal infection. We then compared the results with those of examinations of spinal fluid taken in cases not involving bacterial invasion of the meninges but giving meningeal signs clinically. The results of these studies constitute this report.

TABLE 1.—*Examination of Spinal Fluid in Cases Other Than Meningeal Infections*

Case	Diagnosis	Source	p_H	Carbon Dioxide	Sodium	Potassium	Protein	Calcium	Lactic Acid	Chlorides
1	Nasal sinusitis	B.I.H.*	7.5	74.0	.....	....	...	....	12.1	...
2	Neurologic disorder	B.I.H.	7.5	75.2	311.0	12.0	59	....	9.3	...
3	Chorea	B.I.H.	...	....	.....	....	142	10.1	14.3	...
4	Neurologic disorder	B.I.H.	7.3	74.0	350.7	11.0	81	....	18.0	...
5	Neurologic disorder	B.I.H.	7.4	78.0	342.0	17.1	93	....	16.0	441
6	Diabetes	B.I.H.	7.2	69.0	333.0	10.1	54	....	12.0	439
7	Multiple sclerosis	B.I.H.	...	....	337.0	10.1	59	....	9.4	419
8	Epilepsy	.....	7.0	74.2	330.0	12.0	82	8.4	12.3	...
9	Hyperthyroidism	U.I.Z.†	7.5	73.8	.....	....	62	9.1	10.3	...
10	Nephritis	B.I.H.	7.6	76.1	347.0	11.0	81	9.0	10.1	...
11	Syphilis	B.I.H.	7.5	74.3	.....	....	62	....	9.2	...
12	Gastro-intestinal obstruction	B.I.H.	7.4	69.0	.....	....	85	....	10.4	...
13	Diabetes	B.I.H.	7.6	71.5	.....	....	144	....	21.2	...
14	Pertussis	B.I.H.	7.6	78.1	.....	....	57	8.9	12.1	440
15	Epilepsy	B.I.H.	7.3	97.5	331.6	21.3	117	9.5	24.1	412
16	Pneumonia	B.I.H.	7.2	67.2	331.6	9.8	81	9.1	10.2	443
17	Adenopathy	B.I.H.	7.2	69.3	.....	....	...	....	10.1	431
18	Tabes	.....	7.2	71.1	.....	....	163	....	14.3	414
19	Pneumonia	C.H.‡	7.6	73.6	331.2	12.1	82	9.6	12.2	424
20	Syphilis	B.I.H.	7.4	69.2	341.0	14.0	51	....	12.4	436

* Beth Israel Hospital.
† United Israel-Zion Hospital.
‡ Crown Heights Hospital.

TABLE 2.—*Examination of Spinal Fluid in Cases of Intracranial Lesions not Including Meningitis*

Case	Diagnosis	Source	p_H	Carbon Dioxide	Sodium	Potassium	Protein	Calcium	Lactic Acid	Chlorides
1	Meningism	B.I.H.*	7.0	59.1	331.0	12.1	114	9.1	12.1	419
2	Brain abscess	B.I.H.	7.2	63.0	.....	....	170	....	9.1	420
3	Encephalitis	B.I.H.	7.0	60.1	316.7	....	...	....	....	...
4	Encephalitis	B.I.H.	7.5	72.1	322.0	13.2	231	10.1	10.1	364
5	Encephalitis	B.I.H.	7.4	73.1	322.0	24.0	118	7.7	3.0	432
6	Encephalitis	B.I.H.	7.5	74.1	334.0	12.1	100	9.1	12.1	444
7	Hemiplegia	B.I.H.	7.8	68.7	342.0	....	100	9.4	12.4	426
8	Brain tumor	B.I.H.	7.5	76.1	349.0	11.0	59	9.1	9.1	...
9	Brain tumor	N.I.†	7.6	73.1	356.0	12.0	56	9.1	9.2	...
10	Brain tumor	N.I.	7.5	79.4	320.0	22.7	93.7	9.2	12.1	...
11	Brain tumor	N.I.	7.7	78.8	331.0	12.2	61	10.1	10.2	...
12	Subarachnoid hemorrhage	B.I.H.	7.9	71.7	.....	....	...	....	9.3	...
13	Brain abscess	B.I.H.	7.0	62.0	332.0	20.3	...	10.1	14.1	426
14	Encephalitis	B.I.H.	7.4	68.3	.....	....	71	....	9.2	429

* Beth Israel Hospital.
† Neurological Institute of New York.

In table 1 are recorded our observations in cases which are not intracranial infections. These patients and some normal persons who had lumbar punctures performed for diagnostic purposes serve as controls and establish our "norms."

TABLE 3.—*Examination of Spinal Fluid in Cases of Meningitis*

Case	Diagnosis	Source	p_H	Carbon Dioxide	Sodium	Potassium	Protein	Calcium	Lactic Acid	Chlorides
1	Tuberculous meningitis....	Bd.H.†	6.9	55.7	321.0	24.0	212	9.1	38.1	342
2	Streptococcal meningitis..	Bd.H.	6.7	50.7	330.0	23.0	219	9.5	29.2	368
3	Meningitis*.....	Bd.H.	6.7	45.2	.....	....	...	....	49.4	354
4	Meningitis*.....	Bd.H.	6.8	51.0	331.0	31.4	294	10.2	50.4	361
5	Tuberculous meningitis....	B.I.H.‡	6.9	50.2	281.7	14.2	221	10.1	82.0	354
6	Tuberculous meningitis....	B.I.H.	6.8	46.3	300.0	18.3	226	10.2	88.0	345
7	Tuberculous meningitis....	B.I.H.	6.7	40.1	310.0	23.1	228	10.1	106.0	345
8	Tuberculous meningitis....	B.I.H.	7.1	44.2	293.0	14.7	231	9.6	64.0	382
9	Tuberculous meningitis....	B.I.H.	7.1	44.3	.....	....	...	....	78.0	370
10	Tuberculous meningitis....	B.I.H.	7.1	45.0	.....	....	...	....	84.0	362
11	Tuberculous meningitis....	B.I.H.	7.0	45.0	.....	....	...	....	86.0	368
12	Tuberculous meningitis....	B.I.H.	6.8	49.0	.....	....	212	....	93.0	355
13	Influenzal meningitis.....	B.I.H.	7.0	69.2	331.0	26.0	94	10.1	34.0	372
14	Influenzal meningitis.....	B.I.H.	6.9	65.4	331.0	26.0	106	10.1	39.0	374
15	Influenzal meningitis.....	B.I.H.	6.8	65.1	.....	....	...	....	39.0	368
16	Influenzal meningitis.....	B.I.H.	6.5	62.2	.....	....	...	....	45.0	365
17	Influenzal meningitis.....	B.I.H.	6.8	65.1	.....	....	...	....	52.0	361
18	Influenzal meningitis.....	B.I.H.	6.7	63.1	.....	....	...	....	56.0	367
19	Influenzal meningitis.....	B.I.H.	6.7	62.1	.....	....	...	....	62.0	354
20	Influenzal meningitis.....	B.I.H.	6.7	61.1	.....	....	...	....	64.0	349
21	Influenzal meningitis.....	B.I.H.	6.7	58.4	.....	....	...	....	65.0	354
22	Influenzal meningitis.....	B.I.H.	6.9	60.3	.....	....	...	....	62.0	358
23	Influenzal meningitis.....	B.I.H.	6.9	62.1	.....	....	...	....	64.0	347
24	Influenzal meningitis.....	B.I.H.	6.7	49.2	333.0	29.0	212	....	79.0	344
25	Influenzal meningitis.....	B.I.H.	6.7	48.2	.....	28.0	210	....	81.0	344
26	Meningitis*.....	B.I.H.	6.7	61.0	332.0	26.0	194	....	42.1	381
27	Meningitis*.....	B.I.H.	6.7	43.4	331.0	27.0	198	....	56.4	362
28	Meningitis*.....	B.I.H.	6.7	42.4	.....	....	...	....	64.3	357
29	Meningitis*.....	B.I.H.	6.9	53.8	333.5	27.0	144	10.3	120.0	326
30	Tuberculous meningitis....	B.I.H.	6.7	51.0	331.0	21.0	218	9.6	106.0	364
31	Streptococcus hemolyticus meningitis.....	Bd.H.	6.4	43.1	328.0	24.1	233	9.1	96.0	345
32	Meningicoccal meningitis..	Bd.H.	6.5	47.2	341.0	26.8	264	10.1	82.0	359
33	Streptococcus hemolyticus meningitis.....	Bd.H.	7.0	49.1	341.0	21.2	208	10.2	71.0	364
34	Tuberculous meningitis....	Bd.H.	7.1	49.6	319.0	24.1	218	10.2	47.0	352
35	Meningitis*; mumps.....	B.I.H.	7.0	43.6	314.0	18.0	314	10.2	75.0	...
36	Cerebrospinal meningitis (epidemic).....	Bd.H.	6.8	36.1	334.0	26.0	221	9.1	49.0	362
37	Tuberculous meningitis....	B.I.H.	6.9	43.8	333.6	14.1	144	9.3	34.0	351
38	Tuberculous meningitis....	C.H.§	7.0	57.3	326.0	22.0	210	9.4	51.0	349
39	Streptococcal meningitis..	Bd.H.	6.7	54.2	.....	....	...	....	46.1	364
40	Meningitis*.....	B.I.H.	6.7	61.0	332.0	26.0	194	....	42.1	381
41	Meningitis*.....	Bd.H.	6.3	46.1	332.0	28.0	214	....	29.1	351
42	Meningitis*.....	Bd.H.	6.8	52.1	330.0	23.0	224	....	28.2	364
43	Meningitis*.....	Bd.H.	6.5	50.1	.....	....	164	....	26.1	372
44	Streptococcus hemolyticus meningitis.....	Bd.H.	7.0	52.1	.....	....	181	....	34.1	368
45	Streptococcus hemolyticus meningitis.....	Bd.H.	7.0	54.0	310.5	15.6	104	8.8	33.0	384
46	Tuberculous meningitis....	Bd.H.	6.7	44.0	332.0	21.6	287	9.1	53.0	369
47	Streptococcus hemolyticus meningitis.....	Bd.H.	6.9	49.0	304.7	16.3	208	10.8	22.0.	370
48	Streptococcus hemolyticus meningitis.....	B.I.H.	6.9	50.2	281.7	14.2	221	10.1	82.0	354
49	Tuberculous meningitis....	Bd.H.	6.9	43.4	345.0	21.0	208	10.1	48.2	384
50	Meningococcal meningitis.	Bd.H.	6.7	46.4	341.0	28.0	212	10.0	64.1	333

* Bacterial flora not recorded.
† Board of Health.
‡ Beth Israel Hospital.
§ Crown Heights Hospital.

Table 2 records the chemical changes in the spinal fluid in intracranial lesions which were nonbacterial. None of these patients had meningitis.

Table 3 gives the observations on the spinal fluid in various types of meningitis.[4]

To comprehend the chemical changes which take place in the spinal fluid during the progress of the meningitides it is necessary that the accepted principles of ionic exchange through physiologic membranes and the devices which are known to aid in maintaining the neutrality of the body fluids be understood. Then only can the significance of the deviations from the normal found during the progress of pathologic lesions properly be evaluated and the previously recorded empiric results of many investigators become synthesized into an integral rational whole.[5]

The following facts are at our disposal and have been corroborated by us in a series of over 125 cases.

1. There is more chloride in normal spinal fluid than there is in the blood plasma of the same person. In meningitis this gradient tends to be eliminated and the values found in the spinal fluid and the blood plasma are more nearly alike.

2. In meningitis there is a decrease in the bicarbonate content of the spinal fluid; the value often is found to be as low as 33 per cent below that of the blood plasma.

3. In meningitis the spinal fluid shows a marked increase in lactic acid. The blood plasma in meningitis also shows an increase in its lactic acid content, due probably to the elevation in temperature and other factors of general import, but the increase noted in the spinal fluid is more marked than the increase found in the blood plasma. In other words, the general increase in blood plasma and spinal fluid does not occur proportionately, the rise being most marked in the spinal fluid.

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4. Regarding the laboratory methods used in arriving at the results recorded, it is to be noted that chlorides were determined by the method of Van Slyke, lactic acid by the Clausen method, carbonate by the manometric method of Van Slyke, p_H by the standard hydrogen electromethod and certain of the lower p_H's by the quinhydrone method. Sodium and potassium were determined by the method of Kramer. An excellent critical study of all these methods is given by Peters and Van Slyke (Quantitative Clinical Chemistry, Baltimore, Williams & Wilkins Company, 1932, vol. 2).

5. Kopetzky, S. J., and Fishberg, E.: Changes in Distribution Ratio of Constituents of Blood and Spinal Fluid in Meningitis, J. Lab. & Clin. Med., to be published.

4. The p_H of the spinal fluid in meningitis is much lower than either that of the normal fluid or that of the normal blood plasma; it is lower than the blood plasma in meningeal cases.

A graphic representation of the acid base balance in normal plasma, normal cerebrospinal fluid and the same observations in a case of typical meningitis are shown in chart 1.

The normal spinal fluid is practically protein free, the protein nitrogen ranging from 35 to 100 mg. per hundred cubic centimeters,

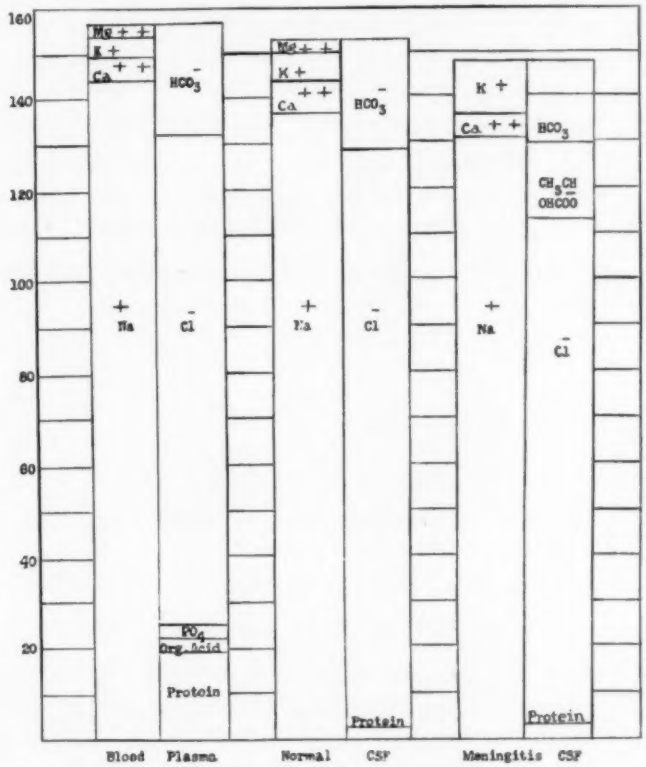

Chart 1.—Acid-base balance in plasma and cerebrospinal fluid in the normal state and in typical meningitis.

while the protein content of the plasma ranges between 6,500 and 8,500 mg. per hundred cubic centimeters. We may, then, think of the dividing line between the plasma and the spinal fluid as one which is permeable to all ions except that of protein. Definite laws govern the ionic interchanges in this type of membrane, which have been summarized by Donnan[6] in his classic work. Let us assume that the

6. Donnan, F. G.: Ztschr. f. Elektrochem. **17**:572, 1911.

membrane is impermeable for the anion P of a salt NaP but permeable for all the other ions, and that the heavy line represents this membrane:

$(\overset{+}{Na})_1$	$(\overset{+}{Na})_2$
$(\overline{Cl})_1$	$(\overline{Cl})_2$
	$(\overline{P})_2$
Spinal Fluid	Blood Plasma

Since osmotic equilibrium must prevail, we can prove by thermodynamic reasoning that

$(Na)_1\ (Cl)_1$ must be equal to $(Na)_2\ (Cl)_2$ and

$$\frac{(Na)_1}{(Na)_2} = \frac{(Cl)_2}{(Cl)_1} = 2$$

where r is a symbol that we adopt to represent the common distribution ratio of the diffusible ions on both sides of the membrane. Let us assume that this represents the conditions on both sides of the membrane dividing the plasma from the spinal fluid. If both the $\overset{+}{Na}$ and $\overline{Cl}$ are diffusible through the membrane we shall have according to Donnan's law

$$\frac{Cl_1}{Cl_2} = \frac{Na_2}{Na_1} = r$$

From chart 2 it can be seen that

$$(Na)_2 = (Cl)_2 + (P)_2$$
$$(Na)_1 = (Cl)_1$$

where $(\overline{P})_2$ is equal to the equivalents of alkali neutralized by the protein of the plasma.

$$\frac{(Cl)}{(Cl)_2 + (P)_2} = \frac{(Na)_2}{(Na)_3}$$

Since the distribution ratio of $(\overset{+}{Na})$ is such that r is practically 1 between the spinal fluid and the plasma, it is apparent that $(Cl)_1$ must be greater than $(Cl)_2$. Such is found to be the case, since the spinal fluid chloride is normally higher than the plasma chloride. With the onset of meningeal infection when there is an increased protein in the spinal fluid, this difference in protein is less, and hence there will not have to be so great a difference between the plasma chloride and the spinal fluid chloride, and the spinal fluid chloride may be even less than in the plasma chloride. This pathologic increase in the protein content of the spinal fluid is, however, not sufficient to account for the great change in the distribution of chloride, and another factor comes into play, which will be discussed later.

For actual clinical use we find that the chlorides in the spinal fluid in meningitis average 350 mg. per hundred cubic centimeters in contra-

distinction to the normal figure of 450 mg. Hence the spinal fluid chloride-plasma chloride gradient drops from 100 to —45. This can be seen in table 4.

The normal lactic acid content of the spinal fluid has been found to be 10 mg. per hundred cubic centimeters, and the normal ratio of the lactic acid in the spinal fluid to the lactic acid in the blood plasma never exceeds 0.6. In certain cases in which, owing to fever, heavy exertion or general circulatory impairment is present, the lactic acid content of the blood plasma is increased and the lactic acid content of the spinal fluid remains normal owing to the absence of specific inflammatory conditions, this ratio may be even lower than 0.60.

It has been shown in animal experiments that by the experimental application of pressure a condition of anemia of the brain and surround-

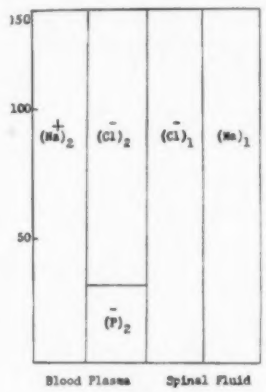

Chart 2.—Approximate ion distribution between the blood plasma and the spinal fluid.

ing tissues may be simulated in which many of the symptoms of meningitis are obtained. It is known that in all conditions in which there is interference with the circulation there is an accumulation of lactic acid. It has been shown by Warburg that in conditions in which there is an insufficiency of oxygen less complete oxidation of carbohydrates takes place and the so-called anaerobic type of oxidation predominates over the aerobic, with the consequent accumulation of lactic acid. The pressure on the blood vessels may be one of the causes of the increased lactic acid in the spinal fluid. Again it is known that the cells of the choroid plexus have a very active glycogen metabolism during embryonic life and also afterward. It is therefore possible that under the influence of certain stimuli they may secrete lactic acid. The old theory that the lactic acid in the spinal fluid is due to the disappearance of the dextrose from the spinal fluid cannot be entirely true because we have found

TABLE 4.—*Observations on Blood and Spinal Fluid in Cases of Meningitis of Various Types Compared with Normal Figures and Those in a Typical Febrile Case (Pneumonia)*

Type of Case	p_H	Carbon Dioxide, per Cent by Volume	Carbon Dioxide Equivalent	Lactic Acid		Protein, Mg. per 100 Cc. Fluid	Sodium, Ce. Tenth-Normal	Sodium, Mg. per 100 Cc.	Potassium		Blood Lactic Acid, Mg. per 100 Cc.	Ratio, Spinal Fluid Lactic Acid/ Blood Lactic Acid	Chloride		Gradient, Spinal Fluid Chloride/ Plasma Chloride	Gradient, Spinal Fluid Carbonate/ Plasma Carbonate
				Mg. per 100 Cc. Fluid	Ce. Tenth, Normal				Ce. Tenth-Normal	Mg. per 100 Cc. Fluid			Mg. per 100 Cc. Fluid	Ce. Tenth-Normal		
Streptococcus hemolyticus meningitis	6.9	40.0	22.2	22	3.6	208	132	304.7	9.0	17	20.9	1.05	350	98.5	—30	—24.0
Streptococcus hemolyticus meningitis	6.9	50.2	22.6	54	6.0	202	122	281.7	7.5	14.2	28	1.92	340	93	—35	— 9.0
Meningitis	6.9	43.8	19.8	120	13.3	144	145	333.5	14.1	27	34	3.54	372	102	—28	—20.1
Meningococcus meningitis	6.9	47.8	21.4	34	3.9	94	137	310.5	11.2	21.3	21	1.62	381	107	—19	—15.2
Tuberculous meningitis	7.1	44.2	19.8	84	9.3	235.6	127	293	14.7	28	28	3.00	362	102	—26	—17.3
Tuberculous meningitis	7.3	51.9	22.9	84.5	9.4	252	137	316	11.2	21.3	26	3.23	354	100	—35	— 9.0
Tuberculous meningitis	7.4	56.6	25.4	84	9.3	267.7	144	332	12.6	24	28.1	2.80	369	102	—24	— 3.5
Meningitis; mumps	7.0	43.6	19.6	75.5	8.4	118	136	314	9.2	18	29	2.60	341	99	—45	—16.2
Meningitis	7.2	50.7	22.7	58.2	6.5	109	137	317	9.6	19	28	2.10	332	93	—46	— 9.4
Meningitis	7.2	50.1	22.6	63	7.0	284	139	320	10.6	21	23	2.70	346	..	—46	— 9.5
Streptococcus hemolyticus meningitis	7.0	40.4	18.2	67.2	7.52	212	134	309	9.2	18	19	3.50	332	93	—47	—21.3
Mastoiditis	7.5	60.0	27.0	21	2.3	...	142	...	...	..	20	1.05	412	116	+17	.....
"Normal"	7.52	63.2	28.5	9	1.0	65*	140	324	9.0	17	14	0.64	442	124	+46	+ 1.2
Pneumonia	7.49	62.1	28.0	18	2.0	...	141.5	329	7.9	15	44	0.41	403	113	+ 3	+ 0.3

* Average normal at Beth Israel Hospital.

cases, as has Killian,[7] in which there was increased lactic acid in the presence of a normal sugar content of the spinal fluid. One of us (Dr. Kopetzky), in his original monograph,[1] reported that in poliomyelitis and meningisms the spinal fluid retained its carbohydrate content and yet showed lactic acid. It has been shown by Levene and Meyer [8] that leukocytes suspended in phosphate solution may give rise to lactic acid as they disintegrate, and some of the lactic acid found in purulent meningitis in the spinal fluid may be accounted for as being formed in the breaking down of the white blood cells. The question of its origin is as yet not entirely settled.

It is, however, well established that there is a rise in lactic acid in the course of meningitis. This was first reported by Glaser [9] and then later confirmed by Killian. Glaser reported the presence of as much as 150 mg. of lactic acid per hundred cubic centimeters in spinal fluids in meningitis. In none of our cases have we found by the Clausen method figures as high as these. We did, however, find variations from double to eight times the normal. In spite of the increased lactic acid content of the blood as a result of the fever in meningitis, there is always a greater increase in the spinal fluid lactic acid. Hence the ratio $\frac{\text{spinal fluid LA}}{\text{plasma LA}}$ is increased. It can be seen that our figures gave a ratio of from 1.5 to 3.5 instead of the normal, which is always less than 1. This ratio gives a good indication, early in the disease, of involvement of the meninges and should be available for diagnostic purposes.

Owing to the accumulation of lactic acid in the spinal fluids there are several interesting consequences which can be predicted on physiochemical reasoning and which we have found to be the case on chemical analysis. The lactic acid is a much stronger acid than carbonic. In the normal spinal fluid the concentration of carbonates, which constitute the main bulwark of the alkaline reserve of the spinal fluid, is slightly higher than the concentration in the blood, averaging about 75 per cent by volume, as determined by the manometric method of Van Slyke. The presence of the stronger lactic acid will tend to displace carbonate from its combination and it will escape as carbon dioxide in the sense of the following equation:

$$NAHCO_3 + CH_3CHOHCOOH = CH_3CHOHCOONa + CO_2 + H_2O$$

Hence we should expect in meningitis to find a lowered carbonate con-

7. Osnato, M., and Killian, J. A.: Significant Chemical Changes in the Spinal Fluid in Meningitis, Arch. Neurol. & Psychiat. **15**:738 (June) 1926.

8. Levene, P., and Meyer, G.: The Action of Leucocytes on Glucose, J. Biol. Chem. **11**:361, 1912.

9. Glaser, J.: The Lactic Acid Content of Spinal Fluid, J. Biol. Chem. **69**: 539, 1926.

tent. This is found to be the case, our values ranging from 35 to 50 per cent by volume. This point is also of value in diagnosing the onset of meningitis.

The presence of the stronger lactic acid should result in a decrease of the alkaline reserve, and hence the spinal fluid should be more acid or, since in the modern nomenclature the actual expression of acidity is the minus logarithm of the actual concentration of the hydrogen ion present, we ought to find a lowering of the p_H. This is found to be the case, and we get values of 6.9 and 7.0 instead of the normal 7.5. When it is borne in mind that a change of 1 in p_H is equivalent to ten times that amount in actual change in concentration of hydrogen ion and that a lowering of the p_H of 0.1 in blood is inconsistent with life, the actual magnitude and fundamental nature of this change become more apparent.

If we were to add 1 cc. of hundredth-normal hydrochloric acid to 1 liter of perfectly pure water of p_H 7.0, the resulting solution would be about p_H 5.0 and would be very toxic to bacteria. If, on the other hand, we were to add the same amount of acid to 1 liter of a standard beef infusion medium of p_H 7.0, the resulting change of p_H would be scarcely appreciable. This power of resistance to change in p_H with added acid was originally likened to the resistance of a "tampon." The word, as used by Sorenson,[10] in its German rendition was *Puffer*, which became in English "buffer." If acid produced by the catabolic processes in the body is added to blood, there is extremely little change in p_H because of the action of the hemoglobin and the serum proteins acting as buffers, which stabilize the reaction. These are missing from the spinal fluid, which is dependent on the carbonates for any real buffer power or ability to resist changes in acid. Since the real stimulus of the respiratory center is due to changes in the p_H content of the body fluid, and a change of 0.1 in p_H has an enormous effect on the stimulation of the respiratory center, it can be appreciated that the need for the sterility of the actual fluid, which probably bathes the respiratory center, is very great. The carbonates function as this buffer. It is known that the maximum buffer efficiency of a mixture of an acid and a salt of this weak acid is attained when this acid and the salt are present in an equimolecular mixture. Henderson has developed an equation for the relation of the p_H and the weak acids and salts which are "buffering" the mixture.

$$p_H = p_K + \log \frac{\text{salt}}{\text{acid}}$$

It is found that the maximum buffer effects are found when salt = acid, and hence the logarithm is 0 and $p_H = p_K$.

10. Sorenson, S. P. L.: Ueber die Messung und Bedeutung der Wasserstaffionenkonzentration bei biologischen Prozessen, Ergebn. d. Physiol. **12**:393, 1912.

In blood the carbonates play a subsidiary rôle in keeping the hydrogen ion concentration stable. There are normally present in the blood about 20 equivalents of sodium bicarbonate but only 1.2 equivalents of carbonic acid. Hence the buffer value must be very low, because its optimum would have to have an equivalent of both the acid and the salt if at maximum. In the spinal fluid, owing to the action of the lactic acid, there are 8 equivalents of carbonic acid released from the carbonate. Hence the ratio, instead of 20:1.2, becomes 12:8.5, which is a much nearer approach to maximum buffer value. The analogy to the low bicarbonate content of the blood and somewhat depressed p_H seen in the terminal stages of diabetic coma due to the accumulation of beta-oxybutyric acid is at once apparent. In the blood plasma, however, there is a fundamental difference because the accumulation of the carbon dioxide at the expense of the plasma bicarbonate results instantly in such a stimulation of the respiratory center that the breathing rate is accelerated, and the blood passes through the lungs at so much greater a rate that the increased oxygenation results in a lower carbon dioxide than normal, so that the buffer action of the carbonates is depressed. With the spinal fluid there is no such passage through an aerating mechanism, and the buffer power of the spinal fluid is increased by the accumulation of the carbon dioxide instead of being depressed by its loss, as in diabetic coma. Expressing this in another form, we have

$$h = \frac{kA}{s}$$

where A is a weak acid, such as carbonic, and s is its alkali salt. We then have in logarithmic form

$$p_H = -\log k - \log A + \log s$$

Hence if we add an amount of acid, such as lactic, to the solution, there is freed from the carbonate an amount of carbonic acid dA and we then have

$$\frac{dA}{dpH} = \frac{-A}{\log e}$$

If we call $\frac{-dA}{dpH} \log e$ the "special buffering power of the solution" B, we then have

$$B = A$$

Hence the buffering power of a mixture of carbonic acid and sodium bicarbonate is independent of the amount of carbonate and proportional to the actual carbon dioxide content. The lactic acid, of course, has no buffer power, since its buffer action is exerted only around a p_H of 4.

The decrease in bicarbonate results in another important consequence. As we have seen in the development of the theory of the Donnan

equilibrium, the anions cross a membrane where they are freely interchangeable. Hence we will have

$$r = \frac{(H\bar{C}O_3)_1}{(H\bar{C}O_3)_2} = \frac{(\bar{C}l)_1}{(Cl)_2}$$

In case of a depression of the bicarbonate due to the accumulation of lactic acid, we would have a momentary state where

$$\frac{(H\bar{C}O_3)_1}{(H\bar{C}O_3)_2} > \frac{(\bar{C}l)_1}{(\bar{C}l)_2}$$

and hence chlorine would have to migrate back into the blood to restore the proper equilibrium. This would result in a lowering of the spinal fluid chlorides, which we have seen is characteristic of meningitis.

It can be seen that there is a definite increase in the potassium content of the meningeal spinal fluid. This is due to the breakdown of the cells, probably mostly the leukocytes. This is an effect that is usually noted accompanying the breakdown of any tissue, because the intracellular tissue fluids are characterized by a greater percentage of potassium than sodium in their composition, in contradistinction to the presence of a greater amount of sodium than potassium in the extracellular body fluids. This has a certain diagnostic value, but the difficulty of determining potassium exactly makes its clinical application difficult.

In conclusion, we submit that the chemical examination of spinal fluid, particularly when the contents are compared to analogous elements in the blood plasma, holds possibilities for early diagnosis of meningitis in clinically obscure cases, and enhances the differential diagnosis of meningeal clinical pictures, when it is necessary to rule out other lesions which simulate the clinical picture of meningitis. Help will be derived from the finding of an increase in the potassium salts, a lower p_H, the lowered carbonate content of the fluid and, more particularly, the marked increase in the lactic acid content of the fluid. In regard to the latter, the marked increase in the ratio of spinal fluid content over the plasma content of the blood will give added diagnostic data.

Clinical Notes

OSTEOMA OF THE FRONTAL SINUS

LOUIS A. PARSELL, M.D., SPOKANE, WASH.

In a survey of the literature inclusive of 1926, Harris [1] reported a total of 117 cases of osteoma of the frontal sinus. I have counted 20 additional cases of osteoma distributed as follows: frontal sinus, 8 cases; orbit, 4 cases; ethmoid, 3 cases; sphenoid, 2 cases, and frontal, ethmoid and orbit, 3 cases.

Osteomas appear to arise from the periosteum lining the sinus. It is doubtful whether trauma plays a rôle. However, there are many instances in which the history of an injury is definite. Chronic inflammation of the sinuses has been suggested as a cause.

Osteomas are usually composed of a dense, ivory-like periphery and a soft cancellous center and are attached to the wall by a pedicle. There are three types: (1) eburnated, (2) spongiose and (3) mixed. Structurally, they differ from the usual bone in that the haversian canals and lacunae do not conform to any definite arrangement and may be absent.

Headache is usually the outstanding symptom of which the patient is aware. The discomfort is usually located in the frontal region. Pain is relatively infrequent and usually occurs in cases in which there is an encroachment on the orbit. Exophthalmos, together with limitation in the movement of the eyes and visual disturbances, accounts for the predominance of cases reported by the ophthalmologist. Deformity is always a late manifestation. The diagnosis is dependent on assistance from the radiologist. The only treatment is complete surgical removal, the usual approach being through a modified Killian incision along the brow. Various modifications of this incision have been suggested. The route pursued should be governed by the size and extent of the tumor.

REPORT OF A CASE

C. G., a man, aged 32, was seen in February, 1932, complaining of right frontal headache and a nasal discharge. The history brought out the fact that the patient was born with a nasal deformity following instrumental delivery, and for several days the nose had been supported by splints. Since childhood he had been subject to frequent colds in the head. In 1918, he began to experience pain in the nape of the neck, which extended from the occiput over the right half of the head. Of late years the headaches had occurred regularly, from two to three times a week, never being associated with nausea or vomiting. His father had died of dementia paralytica at the age of 40. Otherwise, the family history was irrelevant.

I saw the patient first on February 19, when he consulted me because a severe right frontal headache had developed twenty-four hours previously, which had followed an attack of the "flu" two weeks earlier. Examination revealed the deformity, the flattened bridge of the nose and a high deflection to the right of the nasal septum. The left side of the nose was normal. The region over the right frontal

1. Harris, R.: Osteoma of Frontal Sinus, Laryngoscope **38**:331 (May) 1928.

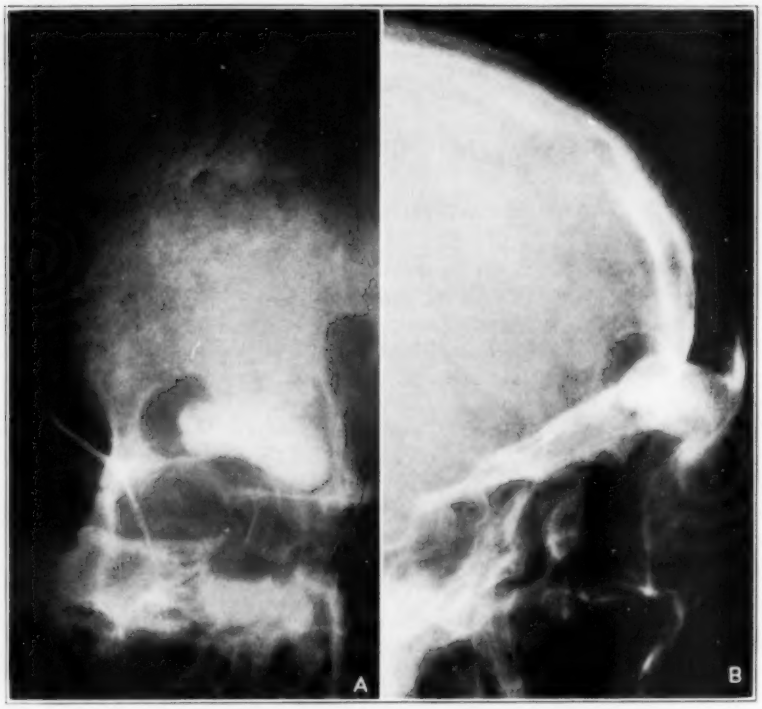

Fig. 1.—*A*, anteroposterior view, demonstrating size and extent of tumor. *B*, lateral view. Note erosion of anterior wall of frontal sinus.

Fig. 2.—Gross appearance of specimen.

sinus was acutely sensitive to pressure. Ocular movements were restricted. The eyegrounds were normal.

The following morning the right eye was completely closed, with edema of the surrounding tissues. There was slight exophthalmos. The headache, however, was less intense, and there was a purulent discharge from the right middle meatus.

On February 22, under local anesthesia, a high submucous resection of the nasal septum was carried out, and the anterior half of the right middle turbinal was amputated. There was no appearance of pus. The anterior ethmoids contained polypi. This region was exenterated.

Subsequently, because the necessity for an external operation seemed imminent, a roentgen examination was made by Dr. Arthur Betts, who reported as follows: "A large osteoma occupies two-thirds of the right frontal sinus. The right ethmoid region is cloudy. The other sinuses appear clear cut."

The patient experienced decided relief following the intranasal operation. In June, he returned, stating that for three days he had been having frequent, sharp, lancinating pains just above the external canthus of the right eye, but no headache. Operation was carried out two days later by Dr. R. T. Atkins.

Under ether anesthesia, with a postnasal plug in situ, an incision along the brow was made, and the periosteum was reflected upward from the supraciliary ridge, exposing the anterior wall of the frontal sinus. The cortex was greatly reduced in thickness, and on removal showed a hard, lobulated tumor mass limited to the frontal sinus but eroding the roof of the orbit. It was attached to the posterior wall by a pedicle. With the aid of a mastoid curet, the osteoma was rocked from its bed and revealed an area of exposed dura measuring 1 cm. in diameter. The dura was covered with granulations. No fistula could be found. The surrounding plate was porous, indicating pressure necrosis, and was removed. The sinus was freely curetted of its lining membrane, which was thickened and covered with multiple small polypi. In separating the tumor from the roof of the orbit, the inner half of the frontal arch was sacrificed. The orbital ethmoids and nasofrontal duct contained polypi. The duct was enlarged, and all of the recesses of the sinus were rounded. A cigaret drain was then introduced into the nose, and additional drains were placed at the inner and outer angles of the wound, which was closed with Michel clips, and a pressure bandage applied. The bony mass measured 3 by 2 by 2 cm.

The cigaret drain was removed after twenty-four hours, and all drainage discontinued on the fourth day. Healing of the wound was without incident. On July 20, the depressed wound was healed, with apparent beginning obliteration of the right frontal sinus. There were slight ptosis of the upper lid, free movement of the globe and no diplopia. There was no discharge from the nose. The patient had gained in weight and had returned to work.

SUMMARY

1. An acute exacerbation of a chronic frontal sinusitis prompted the discovery of the tumor by roentgen examination.

2. Nasal obstruction and susceptibility to colds dated from birth.

3. Intranasal surgical intervention brought relief from headache and cessation of ocular symptoms.

4. A two-stage operation is suggested in the presence of pathologic changes in the nose and accessory sinuses.

Paulsen Medical and Dental Building.

ELECTRICALLY DRIVEN CIRCULAR SAW FOR SECTIONING THYROID CARTILAGE

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One of the most important steps in the performance of a laryngofissure or hemilaryngectomy consists in the sectioning of the thyroid cartilage in the midline. Various means have been advocated for its accomplishment, such as the use of arc-shaped or bayonet-handled saws, Moure shears and the Jackson turbinotome. The use of the saws is slow and awkward, and not infrequently the teeth catch the tissues of the softer intralaryngeal structures, causing them to be torn and shredded. The use of the Moure shears and the Jackson turbinotome is attended by frequent fracturing or splintering of the alae of the thyroid cartilage. These disagreeable accompaniments of the use of the aforementioned instruments are frequently the cause of delayed union, secondary infection, postoperative deformity and laryngeal stenosis. Of course, such accidents are founded on the fact that the thyroid cartilage becomes hard and brittle in later adult life. It has erroneously been assumed in the past that this change in the cartilage was due to a process of calcification. Recently, however, Otto Mayer [1] observed in a histopathologic study of a series of laryngeal perichondritides that the thyroid and cricoid cartilages

Photograph of instrument for sectioning the thyroid cartilage.

became physiologically ossified in the later years of adult life, with the transformation of the cartilage into true spongy bone, containing hematopoietic material and fat marrow. He found that the pathologic picture of inflammation in these structures in that period of life was not that of perichondritis but that of true osteomyelitis.

Mayer's observations offer a reason for the frequent inadequacy of the aforementioned instruments, and it is only natural to turn to accepted instruments for operations on the bones in order to accomplish sectioning of the thyroid cartilage. Consequently, with the cooperation of V. Mueller and Company of Chicago, a small circular electrically driven saw was devised according to the pattern of and not unlike, the well known Albee instrument. The saw is made of extremely thin carbon wafer steel with fine teeth so as to effect a razor-like cut. The blade is 1.87 cm. in diameter and is mounted on a standard shaft to fit the standard handles in use with burrs in surgical procedures on the mastoids and the sinuses (accompanying figure). The use of this saw has been attended by excellent results and satisfaction in the hands of my associate, Dr. Joseph C. Beck, and myself. Splintering or fracturing of the cartilage and tearing or shredding of the soft tissues within the larynx have not been noted, the clear, razor-like incision being made in a few seconds. Postoperative healing is rapid, without any of the disagreeable sequelae previously mentioned.

185 North Wabash Avenue.

1. Mayer, Otto: Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:309 (July) 1931.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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PLASTIC SURGERY

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It is well to be reminded, as one is by Dr. Howard Kelly,[1] of the aids to surgical intervention that science has given in the course of a single lifetime. When the imperative demand came for the covering of defects and the replacement of lost surface tissues, the surgeons of this generation turned to the pages that tell of the work of Szymanowsky, who had done everything, it seemed. But when he lived there was no local anesthesia. Similarly, there has been discovered recently the possibility of removing surface cancers by electric coagulation and excision, the importance of which has been emphasized in these reviews. Whereas the knife leaves the surrounding tissues exposed to infiltration, the electric knife covers them with an impenetrable film; besides, there is no blood to act as carrying agent. One can watch the film for evidence of possible recurrence through failure to eliminate the farthest malignant foci, and can take measures accordingly before a cover is applied. By the time the film disappears, the surgeon feels a degree of security that he has never before experienced. Of course, the debt of plastic surgery to local anesthesia cannot be measured, since it eliminates time as an adverse factor and makes possible the slow, careful suturing that alone enables one to repair a deformity without creating another.

It is true that in respect to another of the advances noted by Dr. Kelly, namely the great aid derived from radium and roentgen therapy in general, the rôle of plastic surgery is, in part at least, that of supplying a remedy for injury done through inability to penetrate the mystery with which the action of the rays is still invested. And yet, the negative is not the only interest, for in dealing, for example, with the edges of a wound after the excision of keloids, it has been found that radium emanations left in place for a short time prepare the tissues for approximation with considerable assurance against the hitherto baffling recurrence.[2]

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1. Kelly, Howard A., and Ward, Grant E.: Electrosurgery, Philadelphia, W. B. Saunders Company, 1932.

2. Sheehan, J. Eastman: A Clinic in Reparative Surgery: Examples of Treatment for Keloids, Unilateral Facial Paralysis and of Various Applications of Skin Grafts, S. Clin. North America **12**:341 (April) 1932.

More immediately pertinent, perhaps, are the experiments being made in relation to the healing of wounds,[3] the emergency treatment of devastating burns,[4] the period that should elapse before the grafting of skin in such burned areas can be efficacious [5] and the disinfection of the skin before incision for reparative purposes. It may be added that, since the behavior of the skin after all precautions are taken against infection arising from conditions in the graft itself may still leave much to be desired, attention is now being given to testing the readiness of the skin to heal by the determination of its oxygen content. In my own service delays decided on with a view to the improvement of such content have proved well worth while. The fact that the healing of wounds depends in part on the reinforcement of the depleted blood supply by the agency of water and salt is so simple and fundamental that its importance might easily be overlooked. The continuing studies on the subject constitute a useful reminder that fundamentals never pass out of the field of investigation and further refinement of details.

The main inference to be drawn from the clinical literature of the year is that the variety of reparative procedures increases in about direct proportion to the intensive effort of recent years. There would have been no article in the literature of twenty years ago presenting as great a number or variety of cases of nasal disfigurement as are now reported by Blair.[6] Neither could there have been such a combination as New and Figi present of cancer, of inability of those who use rays in the treatment of cancer to control this curative medium and of total rhinoplasty by a method in use for not more than five years.[7] That physical afflictions existed in vast numbers during times of peace was always known, of course, but until recently the hopper had no spout. In the matter of nasal deformities, by the way, it is useful, perhaps, to observe that ancient fallacies die hard. The use of ivory in the correction of saddle nose is still practiced,[8] notwithstanding the known intolerance of the tissues for inserted foreign bodies. Whatever temporary tolerance tissues may have for gold, ivory or paraffin, expulsion or worse is ultimately inevitable.

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3. Bird, Clarence E., and Mackay, Eaton M.: The Healing of Wounds, Surg., Gynec. & Obst. **54**:872 (June) 1932.

4. Glover, Donald M.: Six Years of Tannic Acid Treatment of Burns, Surg., Gynec. & Obst. **54**:798 (May) 1932.

5. Blair, V. P.; Brown, J. B., and Haman, W. G.: The Early Care of Burns and the Repair of Their Defects, J. A. M. A. **98**:1355 (April 16) 1932.

6. Blair, Vilray P., and Brown, James Barrett: Nasal Abnormalities, Fancied and Real, Surg., Gynec. & Obst. **54**:797, 1932.

7. New, Gordon B., and Figi, Frederick A.: Use of Pedicled Flaps in Reconstruction of the Nose, Surg., Gynec. & Obst. **53**:780 (Dec.) 1931.

8. Mortnick, W.: Plastic Surgery of the Saddle Nose, Laryngoscope **42**:376, 1932.

There are interesting suggestions of progress in the accounts of individual procedures, as for example the use of full thickness skin grafts to correct deformities [9] of the soft tissues, the use of skin grafts tunneled under the skin after the epithelial layer has been shaved off in aid of tendon repair,[10] the formation of an artificial vagina from a skin graft,[11] the transplantation of tendons [12] and extensive manipulation of the tendons of the hands after a wound.[13] Here again there is evidence of increasing confidence in the behavior and the adaptability of tissues used for replacement in reparative procedures.

Two articles that deal with the grafting principle deserve attention as indicating the extension of that principle to a field in which the utmost delicacy is required. Duel and Sir Charles Ballance have published a description of their method of treating facial paralysis by effecting restoration of the continuity of the nerve.[14] They and Martin [15] have proceeded on the assumption that anastomosis with another nerve will not do, if only because of the inevitable secondary and involuntary energizing of the nerve endings of the anastomosed nerve, with embarrassing arm jerks, etc. Not less interesting, and probably productive of results more gratifying to the majority of such patients, is the method of reanimating the paralyzed facial muscles by pedicle grafts from the temporal and the masseter muscles, with mechanical correction, by means of fascia strips carried from the oral canthal area to the temporal and preauricular areas, of the deformity characteristic of this paralysis.[2]

In closely related fields never entirely separable from the reparative work of the surgeon attention should be directed to Dorrance's monumental discussion of cleft palate [16] and to Veau's new book on the same subject,[17] to Martin's radical method of cheiloplasty after excision

9. Padgett, Earl C.: Full Thickness Skin Graft in the Correction of Soft Tissue Deformities, J. A. M. A. **98**:18 (Jan. 2) 1932.

10. Editorial, Surg., Gynec. & Obst. **54**:117 (Jan.) 1932.

11. Grad, Herman: The Technique of Formation of an Artificial Vagina, Surg., Gynec. & Obst. **54**:200 (Feb.) 1932.

12. Williamson, George A.: Transplantation of Tendons with Stabilization of Paralytic Talipes, Surg., Gynec. & Obst. **54**:953 (June) 1932.

13. Andreasen, Anthony: Section of the Flexor Tendons of the Middle Finger of the Left Hand Through a Palmar Wound, J. Bone & Joint Surg. **14**:700 (July) 1932.

14. Ballance, Sir Charles, and Duel, Arthur B.: Operative Treatment of Facial Paralysis by the Introduction of Nerve Grafts into the Fallopian Canal and by Other Intra-Temporal Methods, Arch. Otolaryng. **15**:1 (Jan.) 1932.

15. Martin, Robert C.: Intra-Temporal Suture of the Facial Nerve, Arch. Otolaryng. **13**:259 (Feb.) 1931.

16. Dorrance, G. M.: Repair of Cleft Palate, Ann. Surg. **95**:641 (May) 1932.

17. Veau, Victor, and Borel, S.: Division palatine, anatomie, chirurgie, phonétique, Paris, Masson & Cie, 1931.

of cancerous tissue [18] and to the recent book by Ivy and Curtis on fracture of the jaws.[19]

Finally, there is an article in the Brussels paper devoted to plastic surgery in which Dantrelle [20] tells of how he, and he was the first in 1916 and 1917 to use this procedure, was led to employ a fusiform full thickness graft from the upper eyelid to cover a defect on another lid. Thus he revealed a source of replacement, not for the lids alone, but for small defects anywhere on the face, provided there are none of those peculiarities of pigmentation to which the lids are susceptible.

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18. Martin, H. E.: Cheiloplasty for Advanced Carcinoma of the Lip, Surg., Gynec. & Obst. **54**:914 (June) 1932.

19. Ivy, Robert B., and Curtis, Lawrence: Fracture of the Jaws, Philadelphia, Lea & Febiger, 1931.

20. Dantrelle: Les greffes de peau totale libres, Rev. de chir. plastique **2**:3 (April) 1932.

Abstracts from Current Literature

Ear

MENINGITIS OF OTITIC ORIGIN. WILLIAM L. RHODES, M. J. & Rec. **136**:5 (July 6) 1932.

A boy, 14 years old, suffered, on December 23, from an attack of otitis media. The condition subsided the following day, and the boy returned to play. Five days later, he complained again of pain in the ear and on December 29 mastoiditis developed on the same side. Operation was performed on the same evening. The mastoid was found to be sclerotic. The antrum and the middle ear were filled with pus, culture of which showed Staphylococcus pyogenes-citreus and Bacillus pyocyaneus. The morning after operation, the patient was conscious and seemed to be in a satisfactory condition. That evening rigidity of the neck developed and spinal puncture showed that the spinal fluid was turbid and under pressure. Examination of the fluid showed a gram-negative diplococcus and a cell count of 4,100. Was this meningitis prior or subsequent to the operation? The author believed that the meningeal invasion was purely of otitic origin, and that it had been in existence several days prior to operation and was not recognized at the time.

WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

VINCENT'S OTITIS MEDIA IN INFANCY. I. NEWTON KUGELMASS, New York State J. Med. **32**:272 (March 1) 1932.

Vincent's infection of one week's duration in a girl, aged 19 months, extended into both ears. Both drums ruptured spontaneously, and Vincent's organisms were found in the discharge from the ears. All local treatment failed to give results. Injections of neoarsphenamine were given once weekly. The nasal accessory sinuses became involved. A double mastoidectomy and adenoidectomy were performed, and the sinuses were treated with packs of solution of mild silver protein. In spite of all treatment, the child returned six months later with a discharge from the right ear, which showed the same infection bacteriologically. The tonsils were then removed.

The author believes that cases of Vincent's infection in the ear are missed because of failure to make bacteriologic examinations of the discharge. As in the case cited they cause irreparable destruction of body tissues.

AIKMAN, Rochester, N. Y. [AM. J. DIS. CHILD.]

THE VESTIBULAR SYNDROME. THE CEREBELLAR SYNDROME. J. A. BARRÉ, Rev. d'oto-neuro-opht. **10**:1 (Jan.) 1932.

Until recent times considered to be Siamese twins, the vestibular and cerebellar syndromes are, nevertheless, often isolated and almost always easily dissociable. Vestibular and cerebellar disturbances may coexist, but it can no longer be maintained that there is a group of symptoms common to both conditions—for example, vertigo, nystagmus and staggering.

The sign of vestibular disharmony, previously described by Barré, is of special interest in cases in which the classical cerebellar signs are not clear. Ordinarily, in a case of vestibular disorder the spontaneous nystagmus is in the direction opposite to the deviation of the arms and trunk. When a cerebellar lesion coexists, the nystagmus is toward the same side as the deviation of the arm, the body deviates to the side opposite to that to which the arms deviate, or deviation of the extended arms is absent even though the Romberg sign is clear and there is

nystagmus. The most frequent cause of vestibular disharmony is a cerebellar disturbance, and this should be first thought of in the absence of a peripheral obstacle (acute otitis supervening on a chronic otorrhea) or of a pyramidal or extrapyramidal lesion. These causes of error are easily recognizable. "Cerebellar nystagmus" and "cerebellar vertigo" have not been considered, because Barré does not believe that there exists a true vertigo or nystagmus without a concomitant vestibular disorder.

The vestibular syndrome is different from the cerebellar syndrome. While the cerebellar signs reveal either asynergia or hyposthenia, the vestibular signs always rest on disturbed tonus, which causes slow deviations of the head, eyes, limbs and trunk. The signs are not unilateral and homolateral for a unilateral lesion; they are bilateral and more marked on the side of the lesion. True vertigo, vestibular nystagmus and slow deviations are the most important signs. With the Romberg test, in vestibular disorders, the deviation of the body is toward the same side as the deviation of the arms and even slight deviations may be measured with the plumb line. It is essential to resort to vestibular tests in cases in which there is no spontaneous nystagmus, vertigo or deviation. All three tests, turning, caloric and galvanic, should be used since they give different results.

Disorders of gait are often notably different in the two syndromes. The vestibular patient may walk correctly, or he may deviate laterally while walking. With a unilateral lesion he usually does not stagger, but deviates to one side when walking forward and to the opposite side on walking backward. Confusion between cerebellar staggering and the abnormal vestibular gait is hardly possible except in cases of intense bilateral vestibular disorder. In cerebellar cases, the difficulty is due to a lack of synergy, to the absence or perturbation of the automatic liaison between the muscles of the trunk and the legs and to dysmetric movements of the segments of the limbs. In vestibular cases, the movements of the segments of the limbs and the trunk are coordinated, but there is a battle between two pulsions acting in opposite directions. The synergy is easily demonstrable in a pure vestibular case by analyzing the movements while the patient is recumbent.

Clinically, a large number of pure vestibular cases are encountered, but the number of pure cerebellar syndromes is small. Difficulty is met when the two are associated, and the error to be avoided is that of attributing to one cause all the signs observed. A number of problems that still need elucidation are suggested. For their solution Barré urges a close cooperation between neurologists, ophthalmologists and otologists, after first agreeing on a uniform technic of examination.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

POSTOPERATIVE SINUS THROMBOSIS. G. KRIEGSMANN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:164 (May 23) 1932.

In a study of hospital records Kriegsmann finds that the sinus was exposed in 183 of 241 antrotomies. Among the 132 cases in which the exposed sinus looked normal during antrotomy there were three cases of secondary sinus thrombosis due to the intervention. The hospital records for radical operation in chronic otitis media showed that the sinus was exposed in 23 out of 86 operations. Secondary sinus thrombosis occurred in 1 of the 18 cases in which the sinus looked normal at exposure. From these observations and a study of the literature the author gained the impression that the deciding factor in the development of secondary thrombosis is the virulence of the etiologic agent. Compression or injury of the sinus may favor the development of a secondary thrombosis, but these cases usually heal without thrombosis, and, on the other hand, thrombosis sometimes occurs in the absence of compression or injury. The author does not advise refraining from exposure of the sinus to decrease the danger of a secondary thrombosis, as this would incur the risk of overlooking a "latent" thrombosis or foci of inflammation in or near the sinus, which the author considers a much greater danger than the rare sinus thrombosis due to surgical exposure; however, he thinks that exposure should be undertaken only

when necessary. He recommends performing antrotomy as late as possible, about three weeks after the beginning of the acute otitis media, to avoid secondary thrombosis. If complications demand early intervention the sinus should be exposed only if there is a positive indication for it; this also applies to operation during an acute exacerbation of chronic otitis, which should be avoided if possible. Kriegsmann advises leaving the retro-auricular wound open following operation if the sinus was exposed. In cases in which a particularly virulent organism is suspected, frequent renewal of tampons and use of a disinfectant on the tampons is recommended.

EDITOR'S ABSTRACT.

TREATMENT OF BRADYECOIA WITH HIGH FREQUENCY SOUND WAVES. E. A. KOPILOWITSCH and M. A. ZUCKERMANN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:208 (May 23) 1932.

Kopilowitsch and Zuckermann have found that treatment with high frequency sound waves (ultrasound) effects an improvement of hearing in certain kinds of bradyecoia manifest in improved tone perception for whispering and for conversation. The best results were obtained in patients with chronic suppurating otitis media with large defects of the tympanic membrane, and in patients with chronic adhesive catarrh when it was not combined with disease of the labyrinth. The distance at which whispering was heard was increased from 30 cm. to 4 meters in patients with chronic suppurating otitis media, and distance for the conversational voice was increased from 25 cm. to 7 meters in 66 per cent of the cases, consisting of several types of bradyecoia. A single good result was obtained in a case of congenital labyrinthine bradyecoia in which the distance at which conversation was heard was increased from 20 cm. to 7 meters in one ear and from 2 to 5 meters in the other ear. Treatment with ultrasound waves effects an improvement of hearing capacity in cases of chronic purulent inflammation of the middle ear which are resistant to all other types of treatment. Little or no improvement in hearing capacity was observed in patients with otosclerosis or in those who had undergone a radical operation. The method used by the authors for the production of the high frequency sound waves and the technic of treatment are described.

EDITOR'S ABSTRACT.

HEALED ABSCESS OF TEMPORAL LOBE AFTER FRACTURE OF PETROUS BONE. W. GRETE, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:245 (June 22) 1932.

Grete reports the case of a man who sustained a T-shaped fracture of the tabular part of the occipital bone and the left parietal bone. On the fifteenth day, slight suppuration of the right ear and a small perforation of the tympanic membrane were observed, and a few weeks later there was more severe suppuration of the ear with simultaneous retro-auricular suppuration. Following incision of the retro-auricular suppuration, there was slight but continuous suppuration from a small fistula, which remained, and from the ear. Several months later a discharge of pus from the retro-auricular fistula occurred during insufflation of the ear. Operation disclosed a large subdural abscess in the right temporal lobe communicating with the antrum and retro-auricularly with the exterior. The operation was followed by complete recovery and restitution of hearing. From a roentgenogram by Stenver's method, the author concluded that a fissure extended from the gaping lambdoid suture to the right mastoid process. The clinical course of the hearing disturbance, a transitory facial paralysis and the absence of vestibular symptoms indicate that the fracture of the pyramid was longitudinal and not transverse. It extended to the tympanic cavity and the auditory meatus, thereby opening paths for the spread of infection. The few analogous cases in the literature indicate that late cerebral abscesses occur chiefly in longitudinal fractures of the pyramid of the petrosa, while late meningitis is more frequently observed after a transverse fracture with fracture of the labyrinth. Exact examination to determine the course of the fracture is important. The develop-

ment of this abscess without any pressure symptoms and without the least impairment of the general condition of the patient illustrates the importance of exact otiatric control of any continuous, fetid suppuration of the ear after fracture of the petrosa. It indicates a complication (extradural, intradural or intralabyrinthine suppuration or a collection of necrotic splinters), which, with the supervention of other seemingly slight symptoms, is an absolute indication for surgical exposure of the fracture.

EDITOR'S ABSTRACT.

PHYSIOLOGY AND PATHOLOGY OF EUSTACHIAN TUBE: NEW METHODS OF STUDY. Á. VON GYERGYAY, Monatschr. f. Ohrenh. **66**:769 (July) 1932.

Von Gyergyay thinks that from an otologic standpoint study of the function of the eustachian tube, which depends on the movement of the muscles and their innervation, is immeasurably more important than the study of morphologic changes of the tube (swelling, constriction). It is on the function of the tube that its penetrability and its opening and closing depend. When continuous air waves capable of being regulated are introduced into the inner ear by means of a special catheter, they favor the opening of the tube and hinder its closing. Observation of the sounds audible in the external auditory meatus during such a procedure led to interesting discoveries. The penetrability of the tube, the strength and direction of movement (opening and closing) and the form of movement differ greatly not only among different persons, but at different times in the same ear of one person. Tones introduced into the nose can be heard objectively in the external auditory meatus when the tube opens, as during yawning or swallowing. The range of tones that penetrate and the strength and color of the tones differ in each person. While it has been thought that the normal tube opens at each yawn and each deglutition, the author finds that in persons with normal hearing movement of the tube accompanies deglutition and yawning only occasionally. Opening of the tube appears to occur when the difference between the air pressure within the tympanic cavity and that outside of it necessitates it. This can occur in blowing the nose and in swallowing, in the resorption of air in the tympanic cavity during corporeal labor, in a change of location, in sudden elevation or in lowering and so on. Serial opening in normal persons occurs when a corresponding difference in air pressure has occurred between the individual deglutitions and yawns, that is, neither too small nor too large. The author has succeeded in changing the healthy tube temporarily into a frequently opening one, by series of deglutitions with an alternately closed and opened nose. His experiments with fatigue and the mechanical effect of the tamponade showed that the too frequent opening of the tube can be artificially moderated. The author has discovered three forms of disease of the tube heretofore unrecognized: that in which the tube opens with each deglutition, that in which the tube is relaxed and incapable of opening in response to swallowing but opens with the slightest air waves introduced by catheter, and that associated with protuberance of the tympanum. The study of the function of the tube and the attempts at increasing the too rare opening of the tube and decreasing the too frequent opening point the way to a rational therapy of diseases of the tube.

EDITOR'S ABSTRACT.

FURTHER INVESTIGATIONS ON THE CORTICAL ARCHITECTURE IN THE DEAF AND DUMB. E. ESCARDÓ and L. HORN, Ztschr. f. d. ges. Neurol. u. Psychiat. **135**:555, 1931.

Horn reported the case of a child, aged 4, who became deaf and dumb following an infection and whose brain on gross examination showed a small supratemporal surface. The Heschl convolution was short and of simple structure, and there was a bilateral loss of the sulcus intermedialis. Microscopically, the cortex of area TB (Economo's classification) was poor in cells, and the cells were smaller than normal. The horizontal and vertical lamination was entirely lacking. The horizontal fibers were destroyed, and there was a lack of radiary fibers. The ves-

tibularis and cochlearis were normal on the left; on the right the cochlearis was completely atrophied. The fibers of the cochlearis in the medulla were normal on both sides and ended in the tuberculum acusticum and neutral cochlear nucleus. Nothing could be determined about the state of these nuclei. In only about one eighth of the cases in the literature have there been found changes in the auditory tract in the deaf and dumb. In the case reported by Horn there were no changes noted. The vestibularis was well supplied with fibers on both sides.

Escardo and Horn report the case of a deaf and dumb child with a deaf and dumb father and a brother who suffered from a similar affliction. The patient died at 41 years of age. The brain showed an excavation of the planum temporale. The sulcus intermedius was lacking on both sides. In the area TB, just as in Horn's case, there was a much less clear striation of the cortex, and the cells were much smaller, changes which are looked on by Escardo and Horn as characteristic of deaf and dumb brains. In addition, the present case shows something which was not found in Horn's first case: much less pronounced granular layers in the cortical area TC. The lack of findings in Heschl's convolutions in the case of Horn was attributed to the early age of the patient at death.

ALPERS, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

Pharynx

EXCHANGES OF CREATININE BODIES IN ADENOIDAL CHILDREN. U. AVELLONE, Pediatria **40**:457 (May 1) 1932.

This paper gives a report of rather detailed study of the relation between the production and the excretion of creatinine bodies in children with hypertrophy of the adenoid tissues. The author found that in these children there is a peculiar pathologic creatinuria concomitant with a diminution of the creatine-creatinine exchange, which becomes modified and much diminished following the removal of the tonsils. It appears that this behavior of the exchange bears some relation to the pharyngeal tonsils and the hypophysis, most probably representing dysfunction of the latter as well as of other glands the function of which depends on it.

SIGNORELLI, New Orleans. [AM. J. DIS. CHILD.]

SURGICAL NARROWING OF PHARYNX FOR CURE OF RHINOLALIA APERTA. A. RÉTHI, Monatschr. f. Ohrenh. **66**:842 (July) 1932.

Réthi says that in a large percentage of cases in which plastic surgery of a cleft palate has been performed there is a persistent rhinolalia which no amount of passive or active exercise will correct. This is due to the shortness of the soft palate and the resulting distance between the lifted velum and the posterior wall of the pharynx. The smaller this gap, the less is the disturbance in speech. The author has devised an operation for narrowing the entrance to the upper part of the pharyngeal cavity. The operation consists in establishing a direct connection between the velum and the posterior wall of the pharynx by joining flaps of mucous membrane from the posterior wall of the pharynx with the mucous membrane of the soft palate. Not only does this result in a considerable narrowing of the passage, but the action of the constrictor pharyngis medius, which ordinarily acts only as a muscle of deglutition, is transmitted to the velum by means of the flap of mucosa which has grown onto the muscle, and thus the latter also takes part in the function of speech. The author gives a description of the technic of this intervention, accompanied by several illustrations, and reports two cases in which it was performed with excellent results. While the operation may be carried out on both sides in one intervention, the author advises an interval of a few weeks. In one case, the improvement in speech after the operation had been performed on one side only was so great that the patient considered operation on the other side unnecessary.

EDITOR'S ABSTRACT.

Larynx

TREATMENT IN A CASE OF STENOSIS OF THE LARYNX AND TRACHEA. O. MAYER, Monatschr. f. Ohrenh. **66**:655 (June) 1932.

Mayer describes his surgical procedure in restoring the speech and the free passage of air in the larynx of a man aged 21 with partial obliteration of the larynx and trachea resulting from diphtheria and tracheotomy at the age of 3. An attempt to close the tracheostoma at the age of 5 was followed by complete occlusion of the larynx and trachea, so that the patient could breathe only through the cannula and spoke with a pharyngeal voice. Surgical intervention revealed great deformity of the cricoid cartilage, absence of the arch and restricted growth of the laminae of the cricoid and the thyroid cartilage. The operation was performed in two stages. The first operation consisted of a combined laryngostomy and tracheostomy, excision of the scars and covering of the resulting defect with skin. A new tracheal tube was thus substituted for the first and second tracheal rings, which were entirely obliterated, and the stenosis below this point was relieved. In the second operation, the opening to the subglottic space was enlarged by cleaving both laminae of the thyroid cartilage from top to bottom by submucous incisions. By this procedure, the transverse constriction due to the acute angle formed by the laminae of the thyroid cartilage as a result of their restricted growth was decreased sufficiently to permit the introduction of a cannula behind the ventral wall of the thyroid cartilage. The cannula was a combination of a chimney-shaped cannula with the speaking cannula of Luer. The author says that resection of the protruded lamina of the cricoid cartilage would not have relieved the stenosis of the subglottic region, which was due to the acute angle of the laminae of the thyroid cartilage, and would have endangered the median fixation of the arytenoid cartilages and thereby the voice. Since recovery from the operation, the patient speaks with a loud resonant voice and also breathes through the larynx, using additional air through the valve only at night or when running. The author discusses the pathogenesis of stenoses of the larynx and trachea resulting from diphtheria in childhood. In this case the anatomicopathologic changes due chiefly to tracheotomy on the one hand and to the diphtheritic process on the other may probably be ascribed in part to daily bouginage with hard rubber bougies after the diphtheritic attack.

EDITOR'S ABSTRACT.

Nose

SUBMUCOUS RESECTION IN CHILDREN. R. E. WINDHAM, Texas State J. Med. **27**:859 (April) 1932.

This is a report of cases of submucous resection in children from 7 to 12 years of age who have been under observation about 6 years without development of nasal deformity. The structures about the nose, i. e., the orbits, the brain and the sinuses, are 95 per cent developed at 7 years of age, and submucous resection after that, when properly done, does not cause malformations. The technic of operative procedure and postoperative care are discussed.

LUECKE, Dallas, Texas. [AM. J. DIS. CHILD.]

TREATMENT OF NASAL INFECTIONS BY THE SUPER-KROMAYER LAMP. EDWARD JAMES DECK, Brit. J. Phys. Med. **7**:6 (April) 1932.

The author describes methods for the treatment of the common cold, chronic nasal catarrh, rhinitis, hay fever, lupus of the nasal mucosa and unhealthy conditions of the nasal cavities existing after the removal of nasal polypi with the Super-Kromayer Lamp (British Hanovia Company). He claims beneficial results.

HARTMAN, Cleveland. [AM. J. DIS. CHILD.]

DEVELOPMENT OF THE FRONTAL SINUS IN THE INFANT AND ITS SURGICAL APPLICATION. R. BOMPET, Rev. med. (Buenos Aires) **17**:623 (Feb.) 1932.

The author believes that the frontal sinus presents certain distinct characteristics in the infant as well as in the adult, the pathologic condition depending somewhat on the age. In the adult the frontal sinus is more frequently affected than the maxillary. This is even more true in the child because of the late formation of the frontal sinus. The origin of the frontal sinus is in the anterior ethmoid cells. In a series of roentgenograms taken on children from 6 months to 18 years of age, the author found that the time of the appearance of the frontal sinus was between the ages of 6 and 8 years. A diagnosis of frontal sinusitis should not therefore be made before the sixth year.

SANFORD, Chicago. [AM. J. DIS. CHILD.]

NASAL DIPHTHERIA IN CHILDREN. SUSANNA GEZELD, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:1 (July 23) 1932.

Gezeld states that the epidemics of diphtheria which occasionally occur in children's wards, especially the contagious wards, usually affect chiefly the nose and are started by the admission of a child with undetected latent nasal diphtheria. Nasal diphtheria occurs most frequently in children under 5 years of age; the largest percentage of cases falls among nurslings. In addition to the typical membranous form, the disease may assume a catarrhal or an erosion form. In the diagnosis of nasal diphtheria, bacteriologic examination should be employed in addition to rhinoscopy. Involvement of the nasal sinuses in cases of diphtheritic infection of the nose is not rare. The duration of the nasal infection depends on the peculiarities of local and general immunity. The author recommends treatment with frequent small doses of antitoxin combined with local therapy.

EDITOR'S ABSTRACT.

IDENTITY OF ELEMENTS OF RESONANCE IN NASAL AND ORAL CAVITIES. A. I. PROSKUROWSKY, Monatschr. f. Ohrenh. **66**:682 (June) 1932.

The analysis of the nasal cavities in their rôle of resonators for the purpose of establishing the identity of their acoustic character with that of the oral cavity forms the subject of Proskurowsky's article. He discusses Helmholtz's resonance theory and Hermann's theory of *formants,* which form, in part, the basis of his study. The author is of the opinion that the process of the formation of sounds in the oral and in the nasal cavities is identical in principle. He discusses the effect of the nasal cavity as a resonator on the formation of the singing voice in the chest and head registers. The theories exposed, he says, bring up the possibility of influencing the change of the mechanism of voice production in the transition to the head register by means of increasing the nasal resonance.

EDITOR'S ABSTRACT.

Miscellaneous

COMMON COLDS AMONG CHILDREN. SAMUEL GOLDBERG, M. J. & Rec. **135**:289 (March 16) 1932.

During the winter one of the greatest problems that face the physician is the prevention and treatment of the so-called common cold. Because colds occur more often in cold weather, the impression prevails that it is the cold weather that transmits this ailment. Accordingly the misguided parent excludes every bit of fresh air from the room and then brings the temperature of the room 15 degrees above normal. On the other hand, there is the parent who follows Gandhi's early teaching regardless of the physical stamina of the child. The common cold is as infectious as any of the infectious diseases. Proper diet, proper elimination from

the bowels and plenty of sunlight, cod liver oil and viosterol build up a resistance against infections. Children's play and rest must be directed. They do not know by instinct, as do animals, when it is time to rest. A consideration of the prevention of cold is frequently a consideration of the prevention of pneumonia.

WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

GOOD COMPENSATION AND ONLY SLIGHT DEFECT OF SPEECH IN UNTREATED CLEFT PALATE. DOUGLAS GUTHRIE, Tr. Med.-Chir. Soc., Edinburgh, 1932, p. 14; in Edinburgh M. J., February, 1932.

A girl, aged 10 years, had not been operated on for cleft palate, though the harelip had been repaired in infancy. During her attendance at the Edinburgh Speech Clinic, her speech was found to be unusually good. The cause was found to be in the closure of the nasopharynx by a mass of adenoid tissue. This is an instance in which it was useful not to remove adenoids.

NEFF, Kansas City, Mo. [AM. J. DIS. CHILD.]

OTORHINOLARYNGOLOGY AND PEDIATRICS. H. ROUECHE, Arch. de méd. d. enf. **35**:450 (Aug.) 1932.

The author stresses the close accord that should obtain between these medical specialties; he enumerates the common problems that present themselves at the bedside, and discusses the interdependence that clinical studies develop. Particularly is this true in cases in which operative procedures may be indicated and in which joint responsibility is assumed by the physician and the surgeon. The particular problems discussed by Roueche include the management of tonsillar tuberculosis, the advisability of tonsillectomy in secondary infections, the treatment of habitual vomiting from stenosis of the esophagus, the differential diagnosis of lymphocytic meningitis following otitis media and tuberculous meningitis and the proper procedure in frank sinus infections in young subjects.

AMESSE, Denver. [AM. J. DIS. CHILD.]

BLOOD SUGAR CURVES FOR RECOGNITION OF LATENT DISTURBANCES IN CARBOHYDRATE METABOLISM IN DRY CATARRH OF UPPER RESPIRATORY TRACT. K. WEINAND, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:42 (July 23) 1932.

Weinand states that primary dry catarrh of the upper respiratory tract may be a symptom of a disturbance of carbohydrate metabolism. If a disturbance of carbohydrate metabolism is at fault, the administration of insulin or, in some cases, merely a low carbohydrate diet usually brings about improvement and cure in a short time. To determine the presence and degree of the disturbance in carbohydrate metabolism, it is necessary to employ intravenous or oral sugar tolerance tests, as a slight or early disturbance of carbohydrate metabolism may escape detection in a single determination of blood sugar. The author gives illustrations of normal and abnormal blood sugar curves following injections of dextrose. If the blood sugar curve is abnormal, antidiabetic treatment should be undertaken; if it is normal, such therapy will be useless in treatment of the catarrh.

EDITOR'S ABSTRACT.

BASIC PRINCIPLES OF VOCAL FATIGUE. H. STERN, Monatschr. f. Ohrenh. **66**:665 (June) 1932.

In addition to the causes for vocal fatigue that have already been established in the literature, Stern points out several others: (1) the use of small or large parts of the vocal musculature for functions for which they were not intended physiologically or in the interest of a voice production conforming to hygienic and esthetic laws; (2) accumulation of products of fatigue in the larynx, especially

in cases in which the latter is inferior; (3) absence of control on the part of the sensory end-organs, which results in lack of proper coordination, and (4) cessation of the finely balanced interplay of acting and counteracting muscles. In some cases, the fatigue can be explained by the development of a static form of work or a form similar to it the main criterion of which is the tremolo. All these factors, which may occur singly, or several of which may be combined, result in manifestations of greater or lesser degree which may, up to a certain point, be designated as miscarried functions of the organs.

EDITOR'S ABSTRACT.

SYMPTOMATOLOGY OF NEOPLASMS OF TIP OF PYRAMID OF PETROSA. V. TANTURRI, Monatschr. f. Ohrenh. **66**:798 (July) 1932.

Tanturri discusses the symptomatology of neoplasms of the tip of the pyramid of the petrosa in connection with the report of three cases. The localization, the point of origin and sometimes the direction of spread can be determined by roentgenologic and clinical symptoms. The author divides tumors of the base of the skull in the region of the tip of the pyramid into three groups according to the location and origin. The first group consists of tumors that do not attack the base of the skull. In pretubal or subtubal origin the neoplasm is objectively demonstrable in the nasopharynx. There is difficulty in swallowing and respiration, and there are otitic symptoms caused by obstruction of the tube. Neuralgia of the trigeminus, especially the third branch, is present. Roentgenography gives negative results. The tumors of the tip of the pyramid that originate in the nasopharynx are characterized by neuralgia of the trigeminus which extends in varying degrees to its three branches; in the beginning it affects the first branch. Paralysis of the external rectus muscle takes place. A retrotubal neoplasm is objectively demonstrable. Otitic symptoms (of the type of secretory catarrh) produced by stasis are present. There is no increase of intracranial pressure. Roentgenography shows erosion of the lower portion of the pyramid. In tumors of the tip of the pyramid of intracranial origin (mesocranial fossa), there is an increase in endocranial pressure. Roentgenography shows erosion in the superior anterior portion of the pyramid. The other symptoms are the same as those of tumors of nasopharyngeal origin, except that the nasopharynx is free from symptoms.

EDITOR'S ABSTRACT.

ALIMENTARY THEORY OF THE PATHOGENESIS OF HAY FEVER. C. E. BENJAMINS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:473 (May 13) 1932.

On the basis of theoretical considerations and results of experiments on guinea-pigs, Benjamins develops a theory of alimentary pathogenesis of grass pollen hay fever. Especially cereals and foods prepared with these are responsible for the development of the hay fever. The sensitization of the hay fever patients is manifest in the positive cutaneous reactions and in the effect of a cereal-free diet. The author reports several cases in which a cereal-free diet was used successfully and also a few failures, in the hope that his colleagues will further investigate the effect of a cereal-free diet on grass pollen hay fever.

EDITOR'S ABSTRACT.

Society Transactions

EDITED by PHILIP E. MELTZER, M.D.

MASSACHUSETTS EYE AND EAR INFIRMARY, OTO-RHINO-LARYNGOLOGICAL SERVICES

Regular Clinical Meetings, Oto-Laryngological Service of H. A. Barnes, M.D. and C. B. Faunce, M.D., Dec. 1 to March 30, 1932

ABSCESS OF THE BRAIN. DR. G. E. SHAMBAUGH, JR., and DR. C. B. FAUNCE.

In a boy, aged 8 years, an acute otitis media developed on the left side four days after the onset of a cold in the head. One week later, he had a convulsion with marked headache and became quite drowsy. On admission, he showed a definite choking of a few diopters of both disks and passed into a deep coma. Intracranial complication was suspected. A simple operation was done on the left mastoid. There was little pathologic process and no evidence of any breaking down. An area of the dura about the size of a silver dollar was exposed, and a temporal decompression was done. A diathermy current was then placed against the dura completely encircling the area of decompression; that is, a ring of diathermy was placed so as to create a walled off area over the region in which an abscess of the brain was thought to be located. The patient was put to bed. Two days later the coma was more marked; the pulse rate had dropped to 60, and he had projectile vomiting. The patient was reoperated on, an incision being made through the dura, which was previously walled off by diathermy, and a huge left temporal lobe abscess was slowly drained. A large-sized Mosher wire basket drain was inserted.

Progress Notes.—Paralysis of the third nerve developed, but all other signs and symptoms excepting the choking of the disks immediately improved. There was no further vomiting, and the pulse and temperature were normal. The wound continued to drain. After three weeks, the wire basket extruded itself spontaneously. There was considerable herniation, which has entirely disappeared. The paralysis of the third nerve slowly improved. Eight weeks after operation, the wound had completely granulated. Except for a slight weakness of the third nerve the child is normal.

ABSCESS OF THE BRAIN. DR. P. E. MELTZER.

A girl, aged 8 years, entered the hospital with an acute exacerbation of a chronic suppurative otitis media on the left side. Her symptoms were those of sepsis—fever, rapid pulse rate and other symptoms. A simple operation was done on the left mastoid, and a sinus phlebitis was discovered. The jugular vein was tied, and the child seemed to get along fairly well except that she had a poor appetite and did not respond as well as would be expected if the sepsis was controlled. A week after the first operation, she had a convulsion, and a slight facial weakness developed on the right side; headache was present. The fundi were normal. There was no evidence of any meningeal irritation. A radical operation was performed on the mastoid, the dura over the tegmen being exposed freely, and an extradural abscess with necrosis over the dura and over the region of the middle ear was found. The margin of the area of decompression was encircled with a diathermy current, and the operation was discontinued. Two days later, a probe was passed through the necrotic area seen at the last operation, and a very foul abscess was drained. A fairly large-sized rubber tube was placed in what appeared to be the abscess cavity.

Progress Notes.—The child continued to improve steadily; all symptoms disappeared, and the appetite improved rapidly. At the end of the third week the drains were extruded, and granulations closed the cavity. At the end of eight weeks, a plastic operation was done and the postaural fistula closed.

DISCUSSION OF CASES OF DRS. SHAMBAUGH, FAUNCE AND MELTZER

DR. H. P. MOSHER: This procedure of encircling the dura with diathermy was presented by Dr. Harold Tobey, who conceived of the idea of using the cutting current to approach the abscess sac. The thing to do is to determine whether or not the diathermy or the coagulating current actually does wall off the arachnoid space so that infected material from the abscess does not leak into the surrounding meningeal spaces.

DR. C. B. FAUNCE: Such an experiment was done; a section of the coagulated dura and necrotic brain tissue was removed and examined by Dr. Ross, who later reported on it.

DR. P. J. ROSS: The section showed an area of markedly thickened dura. There was a dense fibrous tissue which was infiltrated with polymorphonuclears. The pia-arachnoid was agglutinated and adherent to the tissue, both below and above it. It was homogeneous tissue infiltrated with leukocytes. There seemed to be a complete walling off.

DR. L. SCHALL: Is there any way of controlling the degree of heat to be used?

DR. P. E. MELTZER: In using the coagulating current or diathermy, which is a bipolar current, one should use the least amount of heat possible—just enough to create an eschar without actual necrosis. Familiarity and experience with one's electrical apparatus alone can teach one the exact degree of heat. I will say that I have seen motor reactions such as twitching of the face from using diathermy in an area 2 or 3 cm. away from the facial motor area on the cortex.

ABSCESS OF THE BRAIN. DR. G. E. SHAMBAUGH, JR., and DR. P. E. MELTZER.

A girl, aged 13, was admitted to the hospital with the history that three weeks previous to admission an earache developed following extraction of a tooth. The local doctor opened the ear, and it discharged for four days with relief from pain. Immediately following this relief, the girl refused to sit up and lay on the left side with her face pressed against the pillow, complaining bitterly of headache. She became dizzy and vomited if moved. Dr. C. Birdsall, of Haverhill, was called in consultation, and he made a diagnosis of probable cerebellar abscess and sent her to the infirmary. Lumbar puncture gave normal results on admission. There was a spontaneous rotary nystagmus to the left, but no vertigo. She had beginning blurring of the disks and definite adiadokokinesis on the left side and incoordination of the left hand with the pointing test. While under observation by the presenters, her symptoms seemed to improve. She began to vomit again. There was no evidence of any mastoid involvement. Dr. Meltzer performed a simple operation on the mastoid and did not find any evidence of active infection in the mastoid. There was no lead to the dura. He exposed the dura over the cerebellar region for about 3 cm. in all directions and outlined the periphery with diathermy. The operation was stopped. The next day or two vomiting recurred. The cerebellum previously been walled off by diathermy. A Cushing brain needle was inserted; at 4 cm. a definite resistance was felt, and with a little more pressure the needle was then explored. The dura was incised in the center of the space that had entered the abscess cavity. A quantity of pus, probably an ounce or more, was allowed to drain through the needle. A rubber catheter was placed into the abscess. The cerebellum was explored in the region posterior to the lateral sinus. The following day the patient felt much better. The headache disappeared, there was no vomiting, and she had a good appetite for the first time since admission to the hospital. The patient still keeps her head on the left side.

Progress Notes.—The patient continued to show improvement which was interrupted at various intervals of from three to five days by return of vomiting, irritability and loss of appetite but no other signs. On these occasions, without any anesthesia, it was necessary to reexplore the abscess with a Cushing needle, and on every occasion a small amount of pus was drained which would seem to indicate that the catheter did not enter the abscess cavity properly. The depth of the abscess and the lack of symptoms did not warrant any dilation of the established fistulous tract to the abscess. These recurring symptoms lasted three weeks. Finally, the catheter was removed. At this time insertion of the Cushing needle failed to evacuate any pus, but a small amount of thin straw-colored fluid was returned. This ceased after several days; the patient could now move her head freely, and her temperament and disposition were normal.

ABSCESS OF THE BRAIN. DR. G. E. SHAMBAUGH, JR., and DR. C. B. FAUNCE.

A child, aged 5½ years, two weeks before admission had an acute otitis media on the left side with no discharge. The earache cleared, and he seemed quite well until three days before admission to this hospital, when a postaural swelling developed. The local doctor performed an operation on the mastoid. Following the operation, complete motor aphasia developed. The doctor reopened the mastoid and explored the dura; he found considerable granulations over the dura, which he incised. The brain underneath appeared normal, and he did nothing more. The next day the boy was worse, with a temperature of 104 F. There was a paresis of the right hand, right side of the face and shoulder. Dr. C. B. Faunce operated on him and found a very small abscess in the temporal lobe on the left side. A wire basket was inserted, and the child has completely recovered. There is no aphasia or paresis; the temperature is normal, and there is very little drainage from the wound.

DISCUSSION ON CASES OF DRS. SHAMBAUGH, MELTZER AND FAUNCE

DR. C. B. FAUNCE: In the case presented by Dr. Shambaugh and myself, I performed a rather wide decompression and again outlined the periphery of the area of decompression with diathermy. In this case, however, instead of waiting for the usual period of several days to elapse before exploring for the abscess, I decided to explore immediately. Granulations were present all over the tegmen of the dura, in the region of the tegmen of the middle ear and mastoid. I ignored this, however, and made immediate exploration through the walled-off zone in the temporal region. In other words, I ignored the existence of granulations in the undersurface of the temporal lobe and entered directly from the temporal decompression. The culture from this abscess was Streptococcus hemolyticus. In this service we have had three cases of brain abscess due to Streptococcus hemolyticus and one to pneumococcus type III.

We have had an unusual number of cases of brain abscesses in this service in which all but one patient did quite well. The method of drainage has varied from insertion of a rubber drain or catheter into the abscess cavity to repeated puncture of the abscess with the creation of a fistulous tract to the abscess by means of a small rubber catheter. In two cases we have used the Mosher wire basket. The basket has many advantages as well as disadvantages; granulations crowd over the mesh and make it difficult to remove, and the removal is not without danger. On the other hand, it does lend itself nicely to a good inspection of the cavity as well as aids good cleaning and affords good drainage. The rubber tube has been of value in that larger tubes can be inserted, and as large a fistulous tract and lead to the abscess cavity can be maintained as is desired. There is no difficulty in the changing of the tubes. The cannula of the rubber tube or catheter may be obstructive; however, there seems to be ample leakage around it. Repeated tappings of the abscess cavity have in one case proved effective supplemented by the insertion of a small rubber catheter in the tract leading to the abscess. This greatly facilitated insertion of the needle, as it easily slipped

down to the abscess cavity when such treatment was necessary. The one important thing about the handling of these cases has been the lack of trauma to the surrounding tissues. We can reasonably state that the use of diathermy for walling off the meninges about the drainage tract to the abscess has some merit. The procedure of Dr. Harold Tobey of cutting out with endothermy a portion of the dura and brain leading directly down to the abscess sac is of value. There is considerable reaction created by this procedure, but this will be eliminated when we can learn to control our current better. No ill effect has been noted after the transient temperature subsides. There is no immediate urgency for exploration for brain abscess in cases in which the symptoms are not definitely conclusive. One should make a most careful study of the symptoms to aid in localization. The question as to whether or not one should drain through a clean area is still disputable. I believe in entering the abscess through a clean area. One significant symptom in the cerebellar case was the so-called cerebellar posture whereby the head is drawn to the affected side and the eyes are also deviated to that side and occasionally to the opposite side. This girl had a typical cerebellar posture.

Abscess of the Brain. Dr. G. E. Shambaugh, Jr., Dr. C. B. Faunce and Dr. G. Poirier.

A boy, about 7 years of age, five weeks before entering the hospital had an acute otitis media on the left side which discharged for one week. When the discharge stopped he complained of headache, which greatly increased and localized itself behind the left eye. He entered the Massachusetts General Hospital complaining of very severe headache with repeated attacks of vomiting. Here he had an increased spinal pressure of 450 mm., choked disks and increased lymphocyte count of the spinal fluid. Diagnosis of possible brain abscess was made, and he was transferred to the infirmary. While at the hospital, he was quite stuporous, and his pulse rate was as low as 56 at times. A simple operation was done on the mastoid. The mastoid appeared nearly normal, with only a few granulations in the antrum, but nothing more. In view of the fact that there was no history of vomiting or chills, the lateral sinus was not exposed, but the dura was widely exposed over the temporal region; it appeared under increased pressure. The area of decompression was encircled with diathermy, and the next day an incision was made through the dura; exploration was done in all directions to the depth of 4 cm., but no abscess was encountered. Following this exploration, the boy seemed to improve, and his symptoms disappeared after a week. He ate well and slept well. The pulse was not slow. At the end of this time, the pain back of the eye and the headache recurred. Exploration was done by Dr. Poirier in all directions, a Cushing needle being used; no pathologic process was encountered. Neurologic examination that night gave practically negative results. The headache was controlled by acetylsalicylic acid. Ten minutes after the examination, the nurse found the patient dead in bed. Autopsy showed the lateral sinus completely closed off with a firm, white, organized thrombus, but no pus. The surface of the brain showed thrombosis. Small vessels over the temporal region were thrombosed and yellowish; vessels elsewhere were normal. The convulsions were somewhat flattened. The pathologist sectioned the brain and found marked encephalitis. In the center of this central area of softened brain was a firm incapsulated abscess about the size of a golf ball. In this region he found a thrombosed blood vessel, and in following it it led him to another abscess, about 3 cm. from the first one, in the region of the parietal lobe about 6 cm. from the cortex. Both abscesses had a very dense capsule. The culture showed a nonhemolytic streptococcus.

Carcinoma of the Orbit. Dr. H. A. Barnes.

A man, aged about 60, came to the hospital a year previously. At that time he had an extensive carcinoma of the right antrum involving the palate and the malar bone. The ethmoids and sphenoids were also involved. The entire tumor mass was exenterated. No radium was given at that time, but the whole surface was

covered over with coagulating current. I left the service two months later and lost sight of the patient. He now returns to the hospital with a recurrence in the region of the malar bone. At the tumor clinic he was treated by radium and x-rays. I never have seen radium or x-rays have any effect on a massive tumor of that sort. The recurrence was quite extensive. I operated on him again and enucleated the eye, as I wanted him to have every possible chance. I exenterated the orbit, took out the malar bone and went into the temporal fossa; at the same time a large area of dura was exposed. The great wing of the sphenoid was missing and the tumor extended onto the surface of the brain. Herniation was quite marked. All the gross tumor was removed. The dosage is 30 millicurie hours, well screened with gold, and the total dosage will be 5,000 millicurie hours of radiation. So far there has not been the slightest evidence of meningitis.

DR. H. P. MOSHER: Why was the eye not removed at the first operation?

DR. H. A. BARNES: I never remove the eye unless it is absolutely necessary but leave it in for the esthetic result. I have been obliged in some cases to take the eye out later, but I have also seen many eyes that looked as though they should be sacrified, but they were not, and up to this day they have given no evidence of recurrence in the orbit.

DR. H. P. MOSHER: In this case, by leaving the eye there is now an extension into the middle fossa.

DR. H. A. BARNES: To be frank, it was one of the most hopeless cases in which I have ever operated.

DR. GEORGE TOBEY: Does the patient have much pain?

DR. H. A. BARNES: He was having a good deal of pain on admission, but since the operation he is without pain.

SUPPOSED NASAL FIBROMA. DR. G. POIRIER.

The patient was sent from the northern part of New Hampshire for advice concerning the advisability of operation or radium treatment for nasal fibroma. She had two operations, one over a year previously for the removal of polypi and the second one two months previously when some tissue was removed. There was profuse bleeding, and the report from the pathologic laboratory on this tissue was that it showed a juvenile nasal fibroma of the hemangiomatous type. Clinical examination showed that the left side of the nose was filled with polypoid tissue. This mass of tissue extended down into the nasopharynx and bulged the soft palate forward. The roentgen examination by Dr. A. S. MacMillan showed the left ethmoid and antrum completely filled with polypoid membrane. A large mass was made out in the nasopharynx. Nasal fibroma cannot be ruled out. A radical operation on the antrum was done through the canine fossa, and a huge mass of polypoid tissue was removed. A tonsil snare was then passed along the floor of the nose in front of the tumor, which extended into the nasopharynx, and the entire mass was removed. There was no need of any further operation. This morning, in looking into the nose, the region of the middle turbinate can be plainly made out. There are no polypi to be seen anywhere. The nose is clinically clean. Microscopic section of this tissue shows nothing more than a nasal polyp and not fibroma.

DISCUSSION

DR. P. E. MELTZER: Did you do anything to the naso-antral wall surgically, or did the outgrowth of the polypoid tissue erode the naso-antral wall?

DR. G. POIRIER: I removed the naso-antral wall surgically.

DR. HAROLD TOBEY: There were two such cases in the last service. Dr. P. Mysel delivered both of these huge polyps through the naso-pharynx. One originated from the ethmoidal region and the other from the ostium of the sphenoid.

PLASTIC OPERATION ON THE FACE FOLLOWING HEMANGIOMA. DR. ANGUS MCLEOD and DR. V. H. KAZANJIAN.

The patient, a man, was admitted in March, 1928, because of nasal obstruction over a period of from five to six years. He showed at that time a large mass filling the right side of the nose and part of the left, bluish in color and bleeding easily. It was not tender. The mass completely filled the nasal chamber and extended into the nasopharynx. In 1928, Dr. Garland made an external incision through the inner angle of the orbit and the external angle of the mouth, the so-called Moure incision. He exposed a large mass of tissue. It was impossible to remove the tumor completely because of the profuse bleeding. It was necessary to give a transfusion immediately. The patient was discharged three months after operation in a poor physical condition. Three months later he returned because of recurrence of spontaneous hemangioma. Dr. H. Barnes completed the operation, which was extensive removal of the septum, ethmoids and antrum. Because of bleeding, the external carotid was ligated. Nine months later the patient returned to the hospital for a plastic operation. Dr. Kazanjian operated on him; he made a flap on the forehead with the pedicle at the root of the nose. As there appeared to be a recurrence about the face of the sphenoids, a biopsy was done. There was again very free bleeding from this area, which required packing for two weeks. Pneumonia and empyema developed following this packing. Nothing further was done. A year later the man came back and Dr. Kazanjian took the graft which had been prepared for over a year and turned it down to close over the huge external opening in his face. The margins of the skin were freshened and the graft sutured in position.

DISCUSSION

DR. V. H. KAZANJIAN: One can see from the history that the man had a prolonged and stormy time. Wherever the tissue was bare, I grafted skin. There is no evidence of recurrence of the tumor, and the patient is quite a presentable young man.

DR. H. P. MOSHER: This certainly is a very brilliant case, and every one connected with it should be congratulated. I tried to close one or two openings in these cases by working from the cheek. I managed after two or three times to get the openings closed; however, Dr. Kazanjian's method is far better.

DR. H. A. BARNES: I should like to ask if any radium was used.

DR. A. MCLEOD: Yes, it was used the first and second time.

DR. C. B. FAUNCE: Did you use the diathermy?

DR. V. H. KAZANJIAN: Not that I know of.

POUCH OF THE ESOPHAGUS. DR. ROGER RUGGLES and DR. G. POIRIER.

A man, aged 58, came to the hospital complaining of difficulty in swallowing. He had to swallow three or four times before getting the bolus of food down. The roentgenogram showed a fairly large pouch of the esophagus. On February 25 the first stage of the operation was done. A bougie was passed into the esophagus and an incision was made along the sternomastoid muscle. The pouch was identified over the wall of the esophagus; it was dissected free and brought up to the edge of the skin and sewed to the margin with silk. Ten days later the pouch was still attached to the edge of the skin. The adhesions that had formed around it were freed; Dr. Poirier attempted to separate the pouch and was going to suture the raw edge, but the tissues were too friable and it was necessary to tie it off as one would tie off an appendix and invert the stump into the esophagus. Two weeks have now elapsed since the second stage operation, and the patient is swallowing without any difficulty. The fluoroscopic examination with barium sulphate showed the bolus slipping down the entire esophagus without any hesitation.

DISCUSSION

DR. F. GARLAND: In the original operation you identified the pouch but you did not touch it.

DR. R. POIRIER: Yes. This is the fourth case of pouch of the esophagus we have had in this hospital; in one operation was performed by Dr. G. B. Fred with excellent result, in one by Dr. E. W. Herman with partial recurrence of the pouch, in one by Dr. L. Schall with partial success, and in this case up to the present time it seems to be successful.

OSTEOMYELITIS OF THE FRONTAL SINUS. DR. ROGER RUGGLES and DR. H. A. BARNES.

The patient, a boy, entered the hospital with the history that before admission he had a severe attack of headache following a cold which persisted throughout the winter. On entrance a left orbital abscess was present. On the left side of the nose a slight amount of purulent secretion could be seen coming from under the left middle turbinate. The temperature was 101 F. An external incision was made along the nose and the inner canthus of the left eye, and pus was evacuated just over the region of the lacrimal bone. A roentgenogram at this time showed no evidence of any frontal involvement. Because of the acute condition, nothing further was done excepting the establishment of drainage. Three days later, because the swelling of the eye did not recede, another incision was carried further laterally over the eye, and a second pocket of pus was found. Three days following the second operation, he had a sudden rise of temperature and increase in headache and presented signs of meningitis. An operation was done on the frontal and ethmoid sinuses. The signs of meningitis continued, and the patient rapidly became moribund.

DISCUSSION

DR. H. A. BARNES: There was never any evidence of breaking through the floor of the frontal sinus to account for the orbital abscess. The only area of necrosis was from the suture at the junction of the lacrimal and ethmoidal bones. At the third operation in which the frontal sinus was exposed the sinus cavity was examined very thoroughly, particularly the posterior wall. The bone was intact; it looked normal but we felt there was a leak somewhere. At the last operation a wide T incision was made across the entire brow from left to right and then a vertical incision to the hairline from the root of the nose. Flaps were turned back and the frontal areas widely exposed. The posterior wall of the sinus was intact; the mucous membrane was entirely denuded. Biting over the anterior wall of the frontal sinus, as one took a piece of the bone away, pus could be seen leaking from the diploe, forming little lakes at the mouth of each opening. The posterior wall was removed, and large extradural abscesses were found. As we removed the frontal bone we would find multiple areas of extradural abscesses. We continued from the left side over to the right and found a similar condition on the right side. There was at no time any evidence of sinusitis on the right side. The patient's condition required that the operation be stopped. A few days later, with a Hudson drill, multiple openings were made through the frontal bone, and practically the entire frontal bone was removed in an effort to limit the osteomyelitis. As the flap of bone was removed, a large unsuspected extradural abscess was found. It appeared that we got beyond the area of osteomyelitis. With a Cushing needle we explored the frontal lobes; on incising the dura, pus exuded through the puncture, but no definite abscess was found. Since operation the patient has improved. He answers questions rationally. He has a fair memory for members of his family. He knows he has been sick, but he does not remember how long. There is no aphasia or paralysis of any kind.

DR. R. RUGGLES: Lumbar punctures are being done daily. So far there has been no growth until yesterday; that puncture showed Staphylococcus in the spinal fluid. The cell count was 2,000, mostly lymphocytes. Cultures from the orbital abscess, the frontal sinus and the extradural abscess as well as the spinal fluid showed Staphylococcus aureus.

DR. A. S. MACMILLAN: The first roentgenogram does not show any evidence of frontal involvement. The second one three days later shows a definite involvement of the frontal sinus. There is no evidence of osteomyelitis in any of these plates. One would not expect it to show as early as this.

DR. C. B. FAUNCE: I do not understand what the indications were that prompted you to explore the brain for an abscess.

DR. P. E. MELTZER: A considerable number of cases of brain abscess have been found at autopsy which did not present any previous clinical evidence of the condition. Also, a common finding has been thrombosis of the superior longitudinal sinus. There are no localized signs except the loss of memory and change in the character of the patient, and above all there is a certain cerebral look to the patient which we have all seen so many times.

DR. F. GARLAND: I should like to ask if these abscesses have a tendency to come to the surface as one removes the frontal bone.

DR. H. A. BARNES: I can only say that those I have seen have been rather superficial. I believe that in this particular case the infection extended into the meninges by thrombosis of minute vessels and that the drainage of the frontal sinus in no way would influence the end-result. There probably might have been a time when more radical operation might have saved the boy, but I do not think any of us would operate in an acute condition such as this in any radical manner.

Progress Notes.—The patient did poorly. He lived ten days following the last radical procedure. An autopsy was done. On incising the superior longitudinal sinus, it was completely thrombosed for its entire length. Over the surface of both frontal lobes was a thick, creamy, purulent discharge. There was an abscess in the right frontal lobe. The abscess was not encapsulated; there was considerable softening of the brain.

HYPOGLOSSO FACIAL ANASTOMOSIS. DR. H. P. MOSHER.

The patient came into the clinic this morning after a lapse of many years. She had had a radical mastoidectomy which was followed by a facial paralysis. I anastomosed the facial nerve with the hypoglossal nerve. This operation was done in 1914. The facial paralysis was complete. At present, I would consider it an 80 per cent improvement in the function of the nerve. The patient closes her eye quite well. There is still a slight hesitancy on the side of the mouth. Before operation, there was drooling from the corner of the mouth, the lower lid sagged and was constantly tearing.

DISCUSSION

DR. G. TOBEY: How long after the paralysis was the operation performed?

DR. H. P. MOSHER: We waited somewhere between six months to a year. I cannot recollect definitely. This is the second case with improvement—not a very large experience, but satisfactory.

ULCER OF THE ESOPHAGUS. DR. A. S. MACMILLAN and DR. H. P. MOSHER.

Roentgenograms were taken at the Massachusetts General Hospital and showed what was considered to be cardiospasm, that is, marked dilatation of the esophagus down to a point where no barium sulphate was passing through into the stomach. There is also a question of the presence of a malignant condition.

DISCUSSION

DR. H. P. MOSHER: At esophagoscopy, what appeared to be a fibrotic stricture was seen at the lower end of the esophagus. Dilation was done with bougies up to no. 31. The dilation was carried out very cautiously.

DR. A. S. MACMILLAN: Flouroscopy was performed. There was a definite penetrating ulcer of the lower part of the esophagus. The diaphragm was elevated considerably by gas in the stomach, but all films showed a definite defect characteristic of ulcer of the esophagus. It shows on all plates taken. We have had a similar one in the very same location. This is the third case we have had of ulcer of that region that we have been sure of.

DR. H. P. MOSHER: The symptom of spasm is explained easily by the penetrating ulcer that we have seen. I am very much pleased with the outcome in this case.

JUGULAR PHLEBITIS. DR. LEON WHITE, JR.

The patient, a man, aged 28, was admitted to the hospital with a history of acute otitis media following an attack of tonsillitis one month before admission. He states that as a child he had discharge from the ears, but not since then. Examination showed a purulent discharge with sagging of the posterior superior wall of the canal. Roentgenograms showed destruction over the lateral sinus with the stipulation that it was probably an old area of destruction. A simple mastoidectomy was performed on the left side. There was definite infection in the posterior superior canal as well as under the dural plate. No dura was exposed. The lateral sinus was exposed just below the knee and appeared to be thickened. A culture from the mastoid showed short chain streptococcus. The convalescence following the operation was satisfactory for six days, when the patient began to complain of severe headache. The wound was opened to promote better drainage as the healing process had been rapid. The temperature rose to 101.6 F. The manometer test to demonstrate lateral sinus blocking was negative. The spinal fluid showed 270 cells, 95 per cent polymorphonuclears, 5 per cent lymphocytes and no globulin, and there was a trace of sugar. There was no bacteria in the smear. The white blood count was 11,500. These symptoms continued for several days with slight improvement. Four days later, or ten days after the original operation, the patient had a sudden rise in temperature to 104 F. He had no chills, and neurologic signs were negative. The right jugular vein was ligated; the lateral sinus was again exposed and incised, and it bled freely. Blood cultures were negative. Following the jugular ligation and incision of the lateral sinus, the patient's temperature decreased slightly but progressively to remain around 100 F. It is now six days since the ligation of the jugular vein, and the patient shows a smoky urine with considerable albumin and large granular casts.

DISCUSSION

DR. E. CROCKETT: I think it is too early to judge the outcome in this case. I consider a kidney complication a very serious one. In my experience most of these patients did not do well.

DR. H. P. MOSHER: That complication is rare as I remember it. Did you say that after tying the jugular vein the spinal fluid cleared?

DR. L. WHITE: There was a gradual diminution in the number of cells. The last lumbar puncture showed 60 cells, approximately 65 per cent polymorphonuclears and 35 per cent lymphocytes.

Progress Notes.—The patient was referred to the medical department of the Massachusetts General Hospital, and under care continued to show improvement. At the end of three weeks he was practically well. The urine had cleared so that he could go home and be treated as an outpatient. The postoperative wound had entirely healed.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Oct. 3, 1932

CHARLES B. YOUNGER, M.D., *President*

BACTERIOLOGY OF THE MAXILLARY SINUS: CLINICAL ASPECT. DR. OTIS MACLAY.

The purpose of this preliminary study was to determine what organisms are present in chronic infections of the maxillary sinuses, and to make a comparison with those found in the nose. The work extended over about twenty months and included the ordinary infections occurring during the various seasons. The cases were all chronic ones in a quiescent condition, with the exception of two which were subacute. The ages of the patients varied from 12 to 74 years. The history commonly gave a cold or repeated colds or "flu" as the cause of the so-called "head trouble." In three cases infections through the floor of the antrum from infected molars and in one case a chronic hemorrhagic condition were the etiologic factors.

Forty-four cases, occurring in sixty antrums, were studied. In twenty-one cases, involving thirty antrums, a careful comparison of the nasal and antral organisms was made. In the remaining twenty-three, cultures were taken from antral irrigations without consideration of the organisms in the nose. The cultures were obtained under as aseptic conditions as the method allowed.

The appearance of the nasal cavities varied from an absolutely normal picture to one showing pus and polypi. It was extremely interesting, after a careful examination, to find a nose normal in appearance, both before and after shrinking, and still be able to irrigate from the antrum a mass of mucopurulent secretion containing various pathogenic organisms. This justifies the conclusion that sinus infections occur without local clinical manifestations or a history pointing to any local disturbance. In such cases roentgenograms are of great value.

Of the forty-four patients, twenty-one were examined during the year just past (group A) and the remainder during approximately the previous eight months (group B). In thirteen irrigations in group B the return fluid was clear, and in seventeen, cloudy or mucopurulent. Of the clear washings, only one was sterile while three showed no growth from the cloudy or mucopurulent fluid.

Staphylococci were present twelve times in thirty antrums; streptococci, thirteen times, and pneumococci and Hemophilus influenzae, three times. In two cases of group B and in five of group A, Alcaligenes bronchisepticus was found. This organism, isolated first by Ferry in 1910 and later by McGowan in 1911, was considered by them to be the etiologic factor in distemper in dogs. It has been found three times in the nasal secretions from laboratory helpers working with dogs suffering from distemper, but so far as my associates and I have been able to determine has not been reported as coming from the nose or antrum of any one not so associated with the disease. It was found in pure cultures in two cases and in clear washings twice. In cloudy or mucopurulent irrigations it was present four times.

Other organisms not commonly found were those of the colon and proteus groups. In the thirteen clear irrigations from the antrums, staphylococci were present in six and streptococci in five. In the seventeen cloudy or mucopurulent washings, staphylococci were cultivated in six and streptococci in eight.

It is evident from the foregoing observations that the macroscopic appearance of the return fluid does not determine the extent or type of the infection.

In the thirteen nasal cavities in which the antral washings were clear, staphylococci were present eight times and streptococci three times. In the seventeen nasal cavities in which the antral washings were cloudy or mucopurulent, staphylococci were present thirteen times and streptococci, three.

This shows that there were the same number of streptococci in the nasal cavities whether the antral washings were clean or cloudy, and an increase of staphylococci when the antral washings were cloudy. In a decided number of cases the nasal cultures were essentially different from those of the antrums. In others when there was a slight growth of one organism in the nasal cultures there was a heavy growth of the same organism in the antrums. In some cases the nasal and antral cultures were the same, with a heavier growth from the antrum. A variation of the flora of the same antrums at different times occurred. This was shown in the repeat cultures of the Alcaligenes bronchisepticus group. Five of the seven patients returned six months later and showed in the second cultures. with one exception, not only absence of Alcaligenes bronchisepticus, but the presence of various other organisms. This finding was noted in all second cultures. In the one exception the growth of Alcaligenes bronchisepticus was slight the second time and at the third examination was absent.

In the twenty-three cases and thirty antrums in group A, the antrums showed the following organisms, besides those already mentioned: staphylococci, seventeen times, and streptococci, ten times, which in three instances were Streptococcus viridans and in four, Streptococcus haemolyticus. Micrococcus catarrhalis and Proteus vulgaris each appeared once, while Proteus asiaticus was found three times.

These findings demonstrate that severe symptoms are not always associated with the supposedly more virulent organisms. A pure staphylococcic infection may cause marked weakness and inability to make the effort that is essential to success in this strenuous civilization. It is one's duty to find the hidden infection as well as to treat the more readily determined ailments.

In group A only one irrigation returned clear fluid. This is probably explained by the fact that in the more recent work we investigated sinuses in which the history did not point so directly to infection of the sinuses, and from the evidence obtained in the cultural findings and the relief of symptoms the work appears to have been essential. In our future efforts we shall compare the results of the method used in this work with those obtained by the puncture-cannula technic, and also make a careful study of direct smears from the antrum.

BACTERIOLOGY OF THE MAXILLARY SINUS. LABORATORY ASPECT. MRS. RUTH GETTY (by invitation).

The value of the bacteriologic findings in a report of this kind is naturally dependent on the care exercised in isolating and identifying organisms. Therefore, I feel justified in describing the methods used.

Ordinarily from 5 to 15 cc. of antral washings was sent to the laboratory. My associates and I purposely omitted centrifugation of the washings to concentrate the organisms, first, because we felt that any organism which is present in the antrum in sufficient quantity to cause a pathologic condition would be numerous enough to appear in direct culture from the fluid, and second, because we did not want to concentrate possible contaminants from the nose and give them an opportunity to multiply during the delay. Therefore, the cultures were made directly from the fluid as soon as it reached the laboratory.

In each case a freshly poured blood plate was streaked, and a tube of nutrient broth was heavily inoculated. This broth was prepared from fresh beef and contained whatever sugar was present in the muscle.

The plates gave a conception of the numerical relationship of one organism to another, so that we could determine which organism predominated in the antrum. Almost without exception the plates showed the same organism as the broth; however, it made possible immediate isolation for identification. Only organisms that appeared in numerous colonies on the plates were identified and included in the series.

In addition to the study of morphology and appearance on blood plates, the bile solubility test was used as a means of differentiating pneumococci and streptococci.

Gram-negative bacilli were identified by means of fermentation reactions, ability to produce indole, reduction of nitrates, liquefaction of gelatin and motility. Bacillus

influenzae was identified by morphology, failure to grow on any medium which did not contain blood and reduction of nitrates.

Alcaligenes bronchisepticus belongs to the same genus as Alcaligenes abortus and melitensis, the organisms of undulant fever. It has previously been isolated from dogs and cats that presented symptoms of distemper or died of that disease. The organism is pathogenic for rabbits, causing snuffles, and for guinea-pigs, causing respiratory infection. McGowan described a case of irritating chronic nasal catarrh caused by Alcaligenes bronchisepticus in a man constantly handling rabbits and guinea-pigs in the laboratory. The case of a child, aged 5, in whom mild symptoms of pertussis developed twelve days after she had handled a rabbit which had snuffles was reported by Brown. The same organism was isolated from her pet. In a personal communication to Brown, Ferry stated that he and the New York Board of Health Laboratory had recovered the bacillus from the sputum of a laboratory worker who had a typical case of "grip."

Alcaligenes bronchisepticus, isolated in seven of our forty-four cases, was identified by means of the following morphologic and cultural characteristics: It is a very small, gram-negative bacillus, actively motile, usually found singly but occasionally in chains. It grows readily on ordinary mediums in small, rather shiny colonies. Sugars are not fermented, indole is not produced, nitrates are not reduced, and gelatin is not liquefied. Milk becomes alkaline. There is an abundant light brown growth on potato medium.

Of sixty antrums, five, or 8.3 per cent, were sterile; nineteen, or 31.6 per cent, showed pure cultures, and the remaining thirty-six, or 60 per cent, showed a mixed growth.

The organisms found in pure culture were Staphylococcus aureus in eight antrums; Staphylococcus albus in two; Streptococcus haemolyticus in two; Streptococcus viridans in four; Pneumococcus in one, and Alcaligenes bronchisepticus in two.

Staphylococcus (aureus or albus) was present in thirty-seven of the antrums, while Streptococcus (haemolyticus, viridans or nonhaemolyticus) was found in twenty-six. Pneumococcus and Hemophilus influenzae each occurred in only three antrums.

The genus Alcaligenes was recovered seventeen times in all, from nine patients. It is represented by four species, bronchisepticus, bookeri, ammoniagenes and metacaligenes. The last three species are said by Bergey to be isolated from the intestinal tracts. Their pathogenicity for animals is not given.

Proteus asiaticus and the commonly found Proteus vulgaris appeared three and four times respectively. The genus Escherichia, commonly known as the colon group, had three representatives in the series, communior, neopolitana and dubia. Micrococcus catarrhalis was isolated from only one antrum. Spirilla and fusiform bacilli were found once by direct smear.

In the series as a whole, eighteen different species were found, representing nine genuses. We recognize the possibility that some of the closely related organisms may be variants of one organism rather than distinct species. However, we must follow in a study of this type the recognized classification as compiled by Bergey and a committee of the Society of American Bacteriologists. A bacteriologic study of sinuses is not complete unless correlated with pathologic findings. The only means that we have in the laboratory of determining this second factor is examination of cellular contents. In this series microscopic study of sediments was not made in each case. In the future we propose to include a thorough cytologic study of washings from antrums.

DISCUSSION

DR. ALEXANDER DAY: The bacteriologic study of the nose and accessory sinuses is admittedly important as well as interesting. Also, in view of the diversity of reports as to the findings, it is difficult. In 1895, St. Clair Thomson and Hewlett, on the basis of their own investigations in normal persons and of experimental work, concluded that the internal nose in the majority of normal

persons is aseptic, whereas the vestibule is swarming with bacteria. Since that time there have been diverse reports by many persons to the effect that such a conclusion is not warranted and that both the anterior portions of the nose and the fossae are teeming with bacteria. In spite of contradictory findings, the evidence has been steadily accumulating, substantiating the claim of St. Clair Thomson and Hewlett, namely, that the internal nose is relatively free from bacteria.

In considering the nose and accessory sinuses, one must think anatomically of three parts, the vestibule, the fossae and the nasopharynx. Bacteriologically, the flora is very different in the three parts. In the vestibule many bacteria are found and in the fossae relatively few, while in the nasopharynx there are large numbers. What is the mechanism that governs this varying picture? It can be discussed under three heads. First, anatomically there are the mucous membrane of the vestibule and the vibrissae which act as strainers and catch most of the bacteria that are inspired, so that by the time the inspired air reaches the fossae it has been largely cleared of its bacterial content. Again, in the fossae there is this mucous membrane which is extended in area by the turbinate bones. Secondly, there is a mechanical means by which bacteria are removed, that is, through the action of the secretion of the nose and the cilia. Third, there is the bactericidal action of the nasal secretion concerning which, however, experimenters are not in agreement. There is strong evidence that the secretion possesses a bacteriostatic action. Fleming has demonstrated that it contains a lytic agent (lysozyme) capable of destroying certain organisms. The nasal mucosa itself appears to have definite bactericidal properties. It was formerly thought that phagocytosis played an important part. This view has changed, and it is now held that phagocytic action is of minor importance. Many different types of bacteria have been reported in the fossae; they are not constant, varying from time to time in number and kind; many are apparently transient. The number, of course, depends a good deal on the number in the air. An atmosphere heavily laden with bacteria naturally is less susceptible to the straining action of the vestibule. So one cannot speak with certainty of the number on all occasions further than to say that the normal middle portion of the nose is comparatively free from bacteria.

If the foregoing is a picture of the nasal passage itself, what about the antrum? There is, according to the literature, an even greater diversity of reports regarding bacteria in that location. There are some who assert that the normal antrum of Highmore is free from bacteria, and others who maintain that many bacteria are frequently found under normal conditions. What has been said about the flora of the middle portion of the nose has a rather direct application to the flora of the antrum, because it is only a step from the middle portion to the antrum. If it is true, as has been shown experimentally, that the normal antrum is well ventilated, why should not the bacteria occasionally gain access to it? Work done by my associates and me in our laboratories on the middle part of the nose and also on the antrum shows that a large percentage of normal noses have bacteria both in the fossae and to a lesser extent in the antrums. We have found that the organisms in the normal antrum are transient, irregular in occurrence, few and not in any sense of the word constant. In the so-called normal antrum the results of our work are in accord with those reported in the papers read tonight. We are in further accord that in clear washings from the antrum one may find bacteria. The first thing one should question in such an investigation is whether the sample one is dealing with is representative. If what has just been said is true, anything that comes in contact with the nasal passage may become contaminated, but in all of this work one must be practical. The research in our laboratories was done with extreme precaution in obtaining specimens which would probably not be practical in a wide series. Therefore, in evaluating results one must take all of these things into consideration and interpret the findings only on and within the limit of error of the method used.

What is a "normal antrum"? How exact are the methods for making such a determination? Bacteria were found in clear washings from the antrums. Does

that necessarily mean that there was an infection? An infection is defined as a successful invasion of the tissues by bacteria and their successful growth therein. In other words, the mere presence of micro-organisms on the surface does not constitute an infection. One must go beyond the mere finding of organisms in the clear washings to determine the presence of infection. Therefore, I think that the essayists are very wise in going further with this work in order to extend it and to attempt to find out if there is something that they can do with those clear or slightly turbid fluids to detect any further evidence of infection by means of the cell content.

DR. EDWIN MCGINNIS: There has been meat for thought in one point in particular that has been made this evening; namely, that the nasal swab nearly always fails to pick up bacteria that will grow in mediums. All otolaryngologists have the experience of having the patient come in with a big box of tubes and applicators with notations from the laboratory to get some infected material from the various sinuses and areas of the nose; and even though one knows that this patient has an infection in the nasal sinuses and one swabs the membranes in a few days, a report from the laboratory says there is no growth from the swab. Any one who has seen the moving pictures of the ciliary action of the mucous membrane of the middle meatus will realize that the bacteria are "taken for a pretty swift ride" by the cilia from the nose into the pharynx so that swab cultures from the nose are usually of little value.

Dr. Maclay is lucky in having such an efficient co-worker. At the Presbyterian Hospital my associates and I struggled along for years without any bacteriologic service except that given by an intern who did the bacteriologic work. In 1922 and 1923, I fortunately had an intern who was a bacteriologist of note, and he was interested in infections in the nasal sinuses. He studied twenty-five cases bacteriologically and a report of this work was incorporated in an article (McGinnis, E., and Cary, W. E.: *Laryngoscope* **33**:424 [June] 1923). The list of bacteria was comparable to that of Dr. Maclay and Mrs. Getty. We studied the bacteria from the washings of the sinus and then also recovered the bacteria from the mucous membrane of the sinus. Dr. Kistner of Portland did even better than this. He stained the bacteria in situ in the mucous membrane of the maxillary sinus and was able to show by slides the streptococcus in the periosteum, thus proving that the bacteria are not only on the surface but in the membrane itself. We also did some animal experimentation, injecting streptococci and staphylococci into the blood stream of rabbits, guinea-pigs and dogs, and we were able to produce pathologic conditions in the animals' various organs. In cases in which we injected Staphylococcus aureus and Streptococcus haemolyticus we produced interesting disturbances in the chest and heart.

DR. OTIS H. MACLAY: There is no doubt that a great deal remains to be done in this study of the sinuses. It has been interesting, and I expect that the work of the next two years will be even more so. We hope to find out a little more about the subject than we now know. There is one thing that appeals to me outside of the mere bacteriologic side and the technic of getting cultures, and that is that these patients are sick. They come to us and do not appear very ill. However, their efficiency is often reduced to 50 per cent or less of normal. They have seen various physicians, and not much has been done, because the findings are not outstanding. A pathologic condition sufficient to warrant intervention may be present, although no marked reaction is observed. A slight secretion from the nose with a chronically red pharynx and nasopharynx is enough to arouse a suspicion of disease of the sinuses and to prompt a thorough examination. I think that members of the general profession and physicians engaged in the practice of other specialties are not, so to speak, "sinus-minded." When they realize that the tonsils are infected they remove them. Infected teeth are removed also, but they allow the infection of the sinus to go on until it erodes the wall and develops into meningitis. One of the patients reported on here was seen by several physicians; he was told that he had a so-called "catarrh" and advised that if he used an atomizer he would be all right. He had an infection that these physicians did not see, but he was not referred to some one who would be

interested enough to look into the case. I think otolaryngologists should realize that the patients who do not appear sick are often suffering from a chronic infection that is seriously handicapping them. It is one's duty to make a thorough investigation of a patient who does not look very sick and perhaps eliminate future trouble.

REFLEX SYMPTOMS FROM DISEASES OF THE EAR, NOSE AND THROAT. DR. ROBERT SONNENSCHEIN.

Symptoms such as sneezing, cough, tinnitus and pain are not characteristic of any one pathologic entity. Owing to the communications of a number of the cranial nerves, notably the fifth, tenth and ninth, many symptoms due to disease conditions in the throat, mouth, teeth and larynx are referred to the ear, and this is especially true of pain.

Headache as such may be due to conditions in the head itself, such as acute inflammatory conditions in the nose and nasal accessory sinuses, abscess of the septum and furuncles in the vestibulum.

Pain in the ear other than that from acute or chronic infectious processes in the middle ear is often due to a radiation from ulcerative processes in the larynx, especially on the posterior wall, to abscessed or carious teeth or to acute inflammatory processes in and about the faucial tonsils.

Pain in the head may also be due to an inflammatory reaction of the meninges or to acute inflammatory or suppurative conditions involving the cerebrum itself.

Sneezing is an involuntary act due to irritation of the nasal mucous membrane, the afferent impulses being transmitted by filaments of the fifth nerve to the respiratory centers in the medulla and the efferent impulses sent out to the muscles of the chest and larynx.

Tinnitus, or noises in the ear, may be due to local conditions in the external auditory canal, such as plugs of cerumen or foreign bodies, to exudative or other conditions in the middle ear or to some irritative process involving the end-organ of hearing or the auditory nerve itself. Tinnitus may also be due to certain systematic disorders, such as profound anemia, hypertension, low blood pressure and cardiac lesions.

In the literature a considerable number of references to these various symptoms may be found. While a knowledge of this part of the subject is not of any importance in the actual practice of otolaryngology, it is felt by many that an understanding of the historical and general backgrounds of the symptoms in the specialty is from the cultural standpoint highly desirable.

News and Comment

SCIENTIFIC EXHIBIT, MILWAUKEE SESSION OF AMERICAN MEDICAL ASSOCIATION

Application blanks are now available for space in the Scientific Exhibit at the Milwaukee Session of the American Medical Association, June 12 to 16, 1933. The final date for filing such applications is Feb. 13, 1933, after which time assignments of space will be made. The Committee on Scientific Exhibit requires that all applicants fill out the regular application form. Persons desiring to receive application blanks should address a request to the Director, Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago.

AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held in Montreal, Canada, on Sept. 17, 1932, during the meeting of the American Academy of Ophthalmology and Otolaryngology. Seventy-three candidates were examined, sixteen of whom were conditioned or failed.

The Board will hold an examination in Milwaukee on June 12, 1933, during the meeting of the American Medical Association. Application blanks may be secured from the secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha.

H. P. Mosher, M.D., *President.*
W. P. Wherry, M.D., *Secretary-Treasurer.*

CORRECTION

In the September issue of the Archives of Otolaryngology (**16**:422, 1932), in an abstract of an article by Dr. E. Froschels, which appeared in the *Monatschr. f. Ohrenh.* (**66**:454 [April] 1932), the term "bradycardia," instead of "baryecoia," was inadvertently used in the title and also in the body of the article.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Second International Otolaryngological Congress

President: Prof. Dr. A. G. Tapia, Hermosilla, 2, Mod, Madrid, Spain.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde, Heereweg 143, Gronigen, Holland.

FOREIGN

New Zealand British Medical Association, Ear, Nose and Throat Section

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.

Royal Society of Medicine, Section of Laryngology

President: Dr. Walter G. Howarth, 21 Devonshire Place, W., London.
Secretary: Dr. A. Lowndes Yates, 33 Weymouth St., W. 1, London.

Royal Society of Medicine, Section of Otology

President: Dr. Norman Patterson, 82 Portland Pl., W. 1, London.
Secretary: Dr. T. B. Jobson, Netherwood, Epsom Road, Guildford.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Milwaukee. Time: June 12-16, 1933.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Boston. Time, 1933.

American Bronchoscopic Society

President: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Edwin McGinnis, 104 S. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. George Fetterolf, 2010 Spruce St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Washington, D. C. Time: May 9-11, 1933.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Laryngological, Rhinological and Otological Society

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.
Place: Chicago. Time: June 8-10, 1933.

American Otological Society

President: Dr. George L. Tobey, Jr., 270 Commonwealth Ave., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.
Place: Washington, D. C. Time: May 9-11, 1933.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Henry A. Barnes, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. Seventh St., Los Angeles.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.

Southwestern Michigan Triological Society

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Don C. Lyon, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. Harry L. Whitaker, 1612 Fremont St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. M. H. Gill, 36 Pearl St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Gillum, 221 S. Sixth St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. W. J. Marshall, 212 N. Higgins Ave., Missoula.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Time: Semi-annually.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. McWhorter, 106 E. Earle St., Anderson.
Secretary-Treasurer: Dr. G. M. Truluck, Orangeburg.
Time: April and December.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Smart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. Henry Grant Preston, Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. C. Rouglin, 127 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore City Medical Society, Section on Otology and Laryngology

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

Buffalo Oto-Laryngological Society

President: Dr. John F. Healey, 925 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Herrmann E. Bozer, 468 Delaware Ave., Buffalo.
Place: Buffalo Club. Time: Second Tuesday of each month from September to May, inclusive.

Chicago Laryngological and Otological Society

President: Dr. Charles Younger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. Henry M. Goodyear, The Doctors' Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. F. Howland, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Ia.
Time: Third Monday of each month, September to May, inclusive.

Detroit Oto-Laryngological Society

President: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. J. M. Sutherland, 8-257 General Motors Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6.30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. W. Raney, 609 Fannin St., Houston, Texas.
Secretary: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 314 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

Acting President: Dr. D. E. Godwin, 910 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month, September to May, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Edward Leo Berger, 903 Park Pl., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Otolaryngology

Chairman: Dr. Charles Imperatori, 108 E. 38th St., New York.
Secretary: Dr. David H. Jones, 140 E. 54th St., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., third Wednesday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.
Secretary-Treasurer: Dr. Alexander R. Hampsey, 806 May Bldg., Pittsburgh.

Richmond Ophthalmological and Oto-laryngological Society

President: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Charles J. Boehs, 1126 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. L. Scales, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.

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